

**DRAFT**

**RESIDUAL RISK  
REPORT TO CONGRESS**

Office of Air Quality Planning and Standards  
U.S. Environmental Protection Agency  
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## Acronym List

ADI	Acceptable daily intake
AEGL	Acute exposure guidance level
AIHA	American Industrial Hygiene Association
AQUIRE	Aquatic information retrieval
ARE	Acute reference exposure
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAF	Bioaccumulation factor
BMC	Benchmark concentration
BMD	Benchmark dose
CAA	Clean Air Act
CAS	Chemical Abstracts Service
CCME	Canadian Council of Ministers of the Environment
CRARM	Commission on Risk Assessment and Risk Management
CWA	Clean Water Act of 1972
DOE	Department of Energy
DWEL	Drinking water equivalent level
ED <sub>10</sub>	Effective dose at 10 percent response
EHS	Extremely hazardous substance
EPA	Environmental Protection Agency
ERL	Effects Range Low
ERPG	Emergency Response Planning Guidelines
ET	Ecotoxicity threshold
FIRFA	Federal Insecticide, Fungicide, and Rodenticide Act
FQPA	Food Quality Protection Act
GACT	Generally Available Control Technology
GIS	Geographic information system
GLWQI	Great Lakes Water Quality Initiative
HAP	Hazardous air pollutant
HEM	Human Exposure Model
HEAST	Health Effects Assessment Summary Tables
HEC	Human Equivalent Concentration
HI	Hazard index
HRS	Hazard Ranking System
HWIR	Hazardous Waste Identification Rule
ICR	Information Collection Request
IEM	Indirect Exposure Model
IRIS	Integrated Risk Information System
ISCST3	Industrial Source Complex Short-Term 3
K <sub>ow</sub>	Octanol-water partition coefficient
LAER	Lowest achievable emission rate

LC	Lethal concentration
LEC <sub>10</sub>	Lower 95% confidence limit on effective concentration at 10% response
LED <sub>10</sub>	Lower 95% confidence limit on effective dose at 10% response
LOAEL	Lowest-observed-adverse-effect level
LOC	Level of concern
LOEC	Lowest-observed-effect concentration
MACT	Maximum achievable control technology
MCLG	Maximum contaminant level goal
MIR	Maximum individual risk
MOE	Margin of exposure
MRL	Minimum risk level
NAS	National Academy of Sciences
NESHAP	National emission standard for hazardous air pollutants
NOAEL	No-observed-adverse-effect level
NOEC	No-observed-effect concentration
NPL	National Priorities List
NRC	National Research Council
NRDC	Natural Resources Defense Counsel
NTI	National Toxics Inventory
OAQPS	EPA Office of Air Quality Planning and Standards
OERR	EPA Office of Emergency and Remedial Response
ORD	EPA Office of Research and Development
ORNL	Oak Ridge National Laboratories
OSW	EPA Office of Solid Waste
PAH	Polycyclic aromatic hydrocarbon
PAMS	Photochemical Assessment Monitoring Station
PCB	Polychlorinated biphenyl
PIC	Product of incomplete combustion
POM	Polycyclic organic matter
P2	Pollution prevention
QSAR	Quantitative SAR
RAC	Risk Assessment Council
RfC	Reference concentration
RfD	Reference dose
RSC	Relative source contribution
ROC	Reactive organic compound
SAR	Structure-activity relationship
SEQCCS	CCME Subcommittee of Environmental Quality Criteria for Contaminated Sites
SQB	Sediment Quality Benchmark
SQC	Sediment Quality Criteria
TEF	Toxic equivalency factor
TEQ	Toxicity equivalent
TRI	Toxics Release Inventory
TRIM	Total Risk Integrated Model

TRV	Toxicity reference value
UATMP	Urban Air Toxics Monitoring Program
UF	Uncertainty factor
URE	Unit risk estimate

## Executive Summary

Section 112(f) of the Clean Air Act (CAA), as amended, directs EPA to prepare the Residual Risk Report to Congress on the methods to be used to assess the risk remaining (i.e., the **residual risk**) after control technology standards applicable to emission sources of hazardous air pollutants<sup>1</sup> have been promulgated. CAA section 112(f)(1) contains several specific requirements for the report, which are summarized in **Exhibit ES-1**, along with a reference to where each is addressed in the report.

EXHIBIT ES-1 CROSSWALK BETWEEN SECTION 112(f)(1) REQUIREMENTS AND REPORT	
Section 112(f)(1) provision	Discussed in report
112(f)(1)(A) – Methods of calculating the risk to public health remaining, or likely to remain, from sources subject to regulation under section 112 after application of standards	Chapters 3 and 5
112(f)(1)(B) – The public health significance of such estimated remaining risk	Section 4.1.1
112(f)(1)(B) – The technologically and commercially available methods and costs of reducing such risks	Section 4.1.2
112(f)(1)(C) – The actual health effects with respect to persons living in the vicinity of sources	Section 4.2.1
112(f)(1)(C) – Any available epidemiological or other health studies	Section 4.2.1
112(f)(1)(C) – Risks presented by background concentrations of HAPs	Section 4.2.2
112(f)(1)(C) – Uncertainties in risk assessment methodology or other health assessment technique	Section 4.2.3
112(f)(1)(C) – Any negative health or environmental consequences to the community of efforts to reduce such risks	Section 4.2.4
112(f)(1)(D) – Recommendations as to legislation regarding such remaining risk	Section 4.3

Though not specifically required to be included in the Report to Congress, EPA also presents a discussion of its strategy for addressing the requirements under section 112(f)(2) to set additional standards, if necessary, “to prevent an adverse environmental effect.” These standards will consider costs, energy, safety, and other relevant factors. Discussions pertaining to ecological risk assessment are presented in Chapters 3 and 5.

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<sup>1</sup> The Clean Air Act defines hazardous air pollutant as any air pollutant listed under section 112(b), and also provides procedures for adding and deleting pollutants from the list. The terms “**hazardous air pollutants**,” “**HAPs**,” and “**air toxics**” are used throughout this report synonymously to refer to the pollutants listed under section 112(b).

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## **Chapter 1**

This chapter provides a brief introduction to the Residual Risk Report to Congress and describes the scope and organization of the Report. It presents the specific requirements for the Report listed in CAA section 112(f)(1) (see Exhibit ES-1) and briefly discusses each. Chapter 1 concludes with a discussion of peer review in the context of this Report.

## **Chapter 2**

This chapter provides a brief legislative and regulatory background on the CAA air toxics program. Chapter 2 also provides a short history of the development of risk-based programs and of risk assessment as the primary tool used by EPA to analyze the potential impacts of air toxics emissions on the exposed population and environment. Chapter 2 concludes with a discussion of the development of State and local air toxics programs.

### **The pre-1990 legislative approach and strategy of the Air Toxics Program**

The 1970 CAA mandated a health-based program that required EPA to identify and list HAPs based on human health criteria. EPA was to then promulgate standards for each pollutant, on a source category-by-source category basis, at a level that would ensure the protection of public health with “an ample margin of safety.” In the 20 years following the enactment of this legislation, EPA identified eight pollutants as HAPs, and regulated sources of seven of them.

EPA developed a human health risk management framework for setting national emission standards for hazardous air pollutants (NESHAPs) in 1989 and established standards for several source categories of benzene using this approach (EPA 1989b). Under the framework, EPA developed NESHAPs by following two steps: (1) first determine a “safe” or “acceptable risk” level considering only public health factors; and (2) then set an emission standard that provides an “ample margin of safety” to protect public health, considering relevant factors in addition to health, such as costs, economic impacts, technical feasibility, uncertainties, and any other relevant factors.

### **The legislative strategy for air toxics post-1990**

In the 1990 CAA Amendments, Congress shifted the focus from individual pollutants to industrial source categories and developed a phased approach to controlling air toxics emissions. In the first regulatory phase, EPA must promulgate national, technology-based emission standards for source categories emitting any of the 188 listed HAPs above specific emission thresholds. The overall approach is to use available control technologies or work practice changes to get emission reductions in a timely manner for as many of the listed HAPs as possible, regardless of a HAP’s inherent toxicity and potential risk. This technology-based standards program is commonly referred to as the Maximum Achievable Control Technology (MACT) program.

Congress added a human health risk- and adverse environmental effects-based “needs test” in the second regulatory phase. This phase, referred to as “residual risk” standard setting, requires EPA to promulgate additional standards for those source categories that, after imposition of MACT controls, are emitting HAPs at levels that present a potential unacceptable risk to the public or the environment. Congress directed that such residual risk standards should provide an “ample margin of safety to protect public health.” To set more stringent standards to prevent “an adverse environmental effect,” the CAA requires EPA to take into account costs, energy, safety, and other relevant factors.

### **“Ample margin of safety”**

The two-step process culminating with an “ample margin of safety” determination, as established in the 1989 benzene NESHAP and affirmed by Congress in the 1990 CAA Amendments, is the basis of human health risk management decision-making for the residual risk program. The benzene rule preamble describes the first step, in which the acceptable risk is determined, as follows:

The administrator believes that an MIR [maximum individual risk] of approximately 1 in 10 thousand should ordinarily be the upper end of the range of acceptability. As risks increase above this benchmark, they become presumptively less acceptable under section 112, and would be weighed with the other health risk measures and information in making an overall judgment on acceptability. Or, the Agency may find, in a particular case, that a risk that includes MIR less than the presumptively acceptable levels is unacceptable in light of the other health risk factors (EPA 1989b, p. 38045).

The EPA believes that the level of the MIR, the distribution of risks in the exposed population, incidence, the science policy assumptions and uncertainties associated with risk measures, and the weight of evidence that a pollutant is harmful to health are all important factors to be considered in the acceptability judgment (EPA 1989b, p. 38046).

The preamble also states that in the second step, where the standard is set with an ample margin of safety:

EPA strives to provide protection to the greatest number of persons possible to an individual lifetime risk level no higher than approximately 1 in 1 million. In the ample margin decision, the Agency again considers all of the health risk and other health information considered in the first step. Beyond that information, additional factors relating to the appropriate level of control will also be considered, including costs and economic impacts of controls, technological feasibility, uncertainties, and any other relevant factors (EPA 1989b, p. 38046).

Thus, the benzene NESHAP established specific risk management policy for cancer risks, including some numerical criteria. It did not, however, explicitly address non-cancer human health effects or environmental effects.

### **The development of risk assessment methods**

Chapter 2 discusses a number of work products/programs that have shaped the development of risk assessment methods: (1) the National Research Council's (NRC) 1983 report; (2) the NRC's 1994 report; and (3) the Presidential/Congressional Commission on Risk Assessment and Risk Management's (CRARM) 1997 report.

Congress commissioned a report on risk assessment from the NRC of the National Academy of Sciences in the early 1980s. The result was the landmark 1983 study entitled *Risk Assessment in the Federal Government: Managing the Process* (NRC 1983). It described a risk assessment paradigm that included four steps that are integral to any risk assessment: (1) hazard identification; (2) dose-response assessment; (3) exposure assessment; and (4) risk characterization. This paradigm continues to serve as EPA's model for human health risk assessments.

The NRC developed a follow-up report, *Science and Judgment in Risk Assessment* (NRC 1994), mandated by Congress under section 112(o) of the CAA, that examined current risk assessment methods more closely. The 1994 report concluded that EPA should generally retain its conservative, default-based approach to risk assessment for screening analysis in standard-setting; however, the committee made 70 specific recommendations regarding ways the process should be improved. The report also discussed how the risk assessment recommendations could be implemented in the context of CAA section 112, and specifically advocated that EPA develop and use an iterative approach.

Section 303 of the CAA Amendments of 1990 mandated the formation of the CRARM in response to unresolved questions about the approach EPA should take in determining whether any significant risks to human health remain after the implementation of technology-based HAP emission controls under CAA section 112. The CRARM released a final report in 1997. Volume I focuses on the framework for environmental health risk management, and Volume II addresses a variety of technical issues related to risk assessment and risk management, including margin of exposure, margin of protection, management of residual risks from air toxics, comparative risk, bright lines, realistic exposure scenarios, uncertainty analysis, cost-benefit analysis, interagency consistency, and recommendations to specific agencies. The CRARM's framework fosters an integrated approach to addressing complex, real-world issues that affect more than one environmental medium and involve exposures to mixtures of chemicals.

### **The development of ecological risk assessment at EPA**

Ecological risk assessment at EPA began in the 1970s primarily in two program areas, water quality and pesticide registration. In 1986, the Agency published standardized guidelines for deriving water quality criteria and separate standard evaluation procedures for estimating pesticides' effects. By the mid- and late 1980s, EPA recognized a need for consistency in evaluating ecological risks across program offices and a need to make its ecological research efforts more responsive to its risk assessment needs Agency-wide. In 1992, the Agency's Risk Assessment Forum published a *Framework for Ecological Risk Assessment* (EPA 1992a) that could accommodate all the diverse kinds of ecological risk assessments; various efforts to improve ecological risk assessment have followed Agency-wide. In 1996, EPA issued its *Proposed Guidelines for Ecological Risk Assessment* (EPA 1996c) for public comment, which are the basis of the residual risk approach to ecological risk assessment.

### **State and local air toxics programs**

Prior to passage of the CAA Amendments of 1990, the Federal air toxics program progressed slowly. In the absence of a strong Federal program, many State and some local agencies began to respond to the air toxics problem by developing their own programs. Many states in the country currently have an air toxics control program in place addressing, at a minimum, new sources of toxic pollutants. Some have their own regulations that allow them to actively control air toxics emissions to a level protective of human health; others rely on comprehensive policies or authority provided to implement the Federal program. Some programs are risk-based, while others are technology-based.

### **Chapters 3 and 4**

Chapters 3 and 4 directly address the required statutory elements of the report, as shown in Exhibit ES-1. Chapter 3, in response to CAA section 112(f)(1)(A), provides information on the methods for conducting human health and ecological risk assessments for emissions of air toxics, discusses the data required, and describes the methods for evaluating mixtures. Chapter 4 addresses the remaining statutory elements – those listed in CAA sections 112(f)(1)(B), (C), and (D) – in the order listed in the CAA. The contents of Chapters 3 and 4 are summarized below using a question-and-answer format.



**What methods are used in conducting human health risk assessments?** [Section 112(f)(1)(A)]

The following basic steps are integral to human health risk assessments of air toxics.

- ▶ **Hazard Identification:** The first step in a risk assessment is to determine whether the pollutants of concern can be causally linked to the health effects in question. Factors such as the route of exposure, the type and quality of the effects, the biological plausibility of findings, the consistency of findings across studies, and the potential for bioaccumulation all contribute to the strength of the hazard identification statement. Categories of health

effects used or proposed for future use by EPA in hazard identification for air toxics are: (1) chronic non-cancer; (2) acute non-cancer; (3) cancer, with linear dose-response extrapolation; and (4) cancer, with nonlinear dose-response extrapolation.

- ▶ **Dose-response Assessment:** This step is the quantitative characterization of the relationship between the concentration, exposure, or dose of a pollutant and the resultant health effects. When adequate data exist, the typical end product of the dose-response assessment for non-cancer effects is the identification of a sub-threshold dose or exposure level that humans could experience daily for a lifetime without appreciable probability of ill effect. For cancer, the typical goal is estimation of a full dose-response curve.
- ▶ **Exposure Assessment:** EPA's current *Guidelines for Exposure Assessment*, published in 1992, provide the framework for this step. An exposure assessment has four major components: (1) emissions characterization; (2) environmental fate and transport; (3) characterization of the study population; and (4) exposure calculation. Exposure assessments for residual risk from HAPs may include both inhalation and non-inhalation pathways.
- ▶ **Risk Characterization:** This step is where all the information from the previous steps is integrated to describe the outcome of the analysis, and where the uncertainty and variability in the results are described. EPA's 1995 *Guidance for Risk Characterization* is the foundation for this step of the process.



**What is the general approach for ecological risk assessments?** [Section 112(f)(1)(A)]

Ecological risk assessment "evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors." It is a process for organizing and analyzing data, information, assumptions, and uncertainties to evaluate the likelihood of adverse ecological effects. Like health risk assessment, ecological risk assessment gives risk managers an approach for considering available scientific information along with the other factors they need to consider (e.g., social, legal, political, economic) in selecting a course of action. As defined in EPA's *Proposed Guidelines for Ecological Risk Assessment*, ecological risk assessment consists of three primary phases:

- ▶ **Problem Formulation:** Important steps include identifying assessment endpoints, developing the conceptual model, and preparing an analysis plan.
- ▶ **Analysis:** This phase involves evaluating exposure to stressors and the relationship between stressor levels and ecological effects.
- ▶ **Risk Characterization:** The risk is estimated through integration of the exposure and ecological effects assessments.



**What data are needed for conducting risk assessments?** [Section 112(f)(1)(A)]

Regardless of the endpoint of interest (acute and chronic non-cancer, cancer, or ecological effects), consensus toxicity values are preferred for conducting risk assessments. For chronic non-cancer and cancer values, the preferred source of data is EPA's Integrated Risk Information System (IRIS). Other chronic consensus toxicity values that have undergone less rigorous internal Agency review are available in HEAST, the Health Effects Assessment Summary Tables. For ecological risk assessments, a hierarchy of preferred data sources is more difficult to identify and depends on the type of assessment (e.g., screening versus refined, type of ecosystem at risk). EPA plans to establish data source hierarchies for each type of toxicity information to be used in residual risk assessments.

The focus and level of detail involved in characterizing emissions, and thus the data needs, depend on the nature of the source category and the scope and depth of the overall risk assessment. Dispersion models, which have varying input data needs depending on complexity, are generally used to estimate ambient air concentrations from HAP emissions, while various multimedia fate and transport models are used to estimate HAP concentrations in soil, water, sediment, and biota. Ambient concentrations can also be estimated geographically by monitoring, although the interpretation or use of monitoring data in risk assessment may be confounded by a number of factors. Various other data, including population and ecological receptor information, are also needed to assess exposures.



**What is EPA's general framework and rationale for determining the public health significance of risks remaining after application of a MACT standard to a source category?** [Section 112(f)(1)(B)]

The general framework is based on the risk management approach established in EPA's NESHAP for benzene, which was promulgated in 1989. Congress said in the 1990 CAA Amendments that risk management under the residual risk program should conform with the risk management approach in the pre-1990 version of the CAA, and specifically referred to the 1989 benzene NESHAP.



**What specific criteria does EPA intend to use to evaluate public health significance in the residual risk program?** [Section 112(f)(1)(B)]

EPA plans to use separate criteria to evaluate the public health significance of screening-level and refined risk assessment results. Exhibit 14 in the report (Section 4.1.1) summarizes the criteria for carcinogens for screening and refined analyses; the criteria for non-cancer effects are still under development. With regard to the screening analysis, EPA will consider a range of available toxicity values and simple exposure modeling approaches in determining if the continued emission of HAPs poses a risk to public health or the environment. The refined analysis will be used to more specifically determine whether HAP emissions from a source category pose a continued risk to public health or the environment and whether additional

emission reductions are needed. The refined risk assessment reduces the level of uncertainty by requiring that EPA consensus toxicity values, or equivalent, be used and that more refined estimates of human exposure and uncertainty be developed. This requirement ensures that toxicity criteria of consistently high quality and derived by a consistent methodology are used in the assessment.



**What are the available methods and costs of reducing residual risks?** [Section 112(f)(1)(B)]

Requiring MACT on major sources does not necessarily guarantee that HAP emissions will be reduced sufficiently to protect public health. The CAA requires EPA to promulgate additional standards beyond MACT for major sources of HAPs if required to “provide an ample margin of safety to protect public health.” EPA believes that for most source categories there are reasonable options beyond MACT – including pollution prevention methods – if it is determined that additional emissions reduction is needed. Given the timing of this report in relation to MACT standard implementation and residual risk analyses, it is not possible to determine the specific methods or estimate the costs to reduce residual risks.



**What is the current state of knowledge regarding actual health effects of HAPs on humans based on epidemiological, laboratory, and other exposure studies?** [Section 112(f)(1)(C)]

Information available on actual health effects resulting from exposures to air toxics is limited. Most health effects studies do not focus on populations near sources of HAPs, and information on potential health effects of air toxics is primarily based on laboratory animal and occupational studies. Animal studies suggest potential adverse effects, but usually evaluate chemicals at higher exposures than normally expected for human populations. Occupational human data give evidence of potential effects, but are often limited by a lack of clarity about actual exposure conditions and the fact that occupational exposures are typically higher than those resulting from the ambient air. This Report presents a summary discussion of epidemiological data, laboratory data, and other exposure study data. It also briefly describes how EPA intends to use these data and any actual source category-specific health effects data that may become available when residual risk assessments are conducted.



**What is EPA’s strategy for collecting and assessing epidemiological and actual health effects data?** [Section 112(f)(1)(C)]

EPA recognizes the difficulties that exist in obtaining actual health effects data and conducting epidemiological studies. However, EPA believes that it is useful to incorporate any available health effects/epidemiology data in the residual risk assessments for selected air pollutants and source categories and intends to use such data wherever possible in the decision-making.

In the data gathering stage, EPA will search the scientific literature for published epidemiological studies related to the specific source categories, HAPs, and/or locations studied. Where published epidemiological studies are unavailable, EPA will consider examining other human health data for confirmation of correlations between exposure and adverse effects. However, EPA expects that such data will rarely be available.

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**What is EPA's general strategy on background concentrations?** [Section 112(f)(1)(C)]

Background concentrations are defined generally as the levels of contaminants that would be present in the absence of source-related contaminant releases. Background concentrations come from either contaminants that may occur naturally in the environment or contaminants that are emitted by other (i.e., not the sources being assessed) anthropogenic sources. Narrowly defined for HAPs and the residual risk program, background concentrations are the levels of HAPs in environmental media that are attributable to natural and anthropogenic sources other than the source under evaluation.

At this date, EPA does not have comprehensive Agency-wide guidance or policies on incorporating background concentrations into risk assessments and risk management decisions. Furthermore, analyses of background concentrations and risks can be extremely data- and resource-intensive. EPA's general approach in previous risk assessments and risk management decisions has been to assess incremental risk of a particular source or activity and compare that risk to an acceptable risk criterion. The residual risk program will continue to use this approach, although background concentrations may be considered in the refined analysis for some source categories.

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**What is EPA's approach to addressing uncertainty and variability in the estimation of residual risks?** [Section 112(f)(1)(C)]

The Agency has published several guidance documents addressing this issue, as well as a recently released revision of the *Exposure Factors Handbook* that supports probabilistic approaches to the treatment of a number of commonly employed human health risk assessment input variables. While the exact approach to be taken has not been finalized and may differ from source category to source category, a number of general approaches may be considered for addressing uncertainty and variability in residual risk assessments, including: (1) qualitative assessment; (2) multi-scenario approaches and limited sensitivity analysis; (3) systematic sensitivity analysis; and (4) Monte Carlo simulation and related probabilistic methods.

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**How will EPA meet the requirement to investigate and report on any negative health or environmental consequences to the community of efforts to reduce residual risks?** [Section 112(f)(1)(C)]

EPA recognizes the possibility of creating or transferring risks as an unintended by-product of actions that may be taken to reduce residual risks of HAPs. EPA intends, as part of the section 112(f) standard-setting process, to identify potential negative health and environmental consequences and consider the risk-risk tradeoffs associated with any standards established under the residual risk program. Where deemed necessary, EPA will conduct analyses of these tradeoffs at an appropriate level of detail.



**Is EPA recommending legislative changes to Congress in this report?** [Section 112(f)(1)(D)]

No. At this time, EPA believes that the legislative strategy embodied in the 1990 CAA Amendments adequately maintains the goal of protecting public health and the environment and provides a comprehensive and flexible strategy for addressing a variety of air toxics risk problems.

## ***Chapter 5***

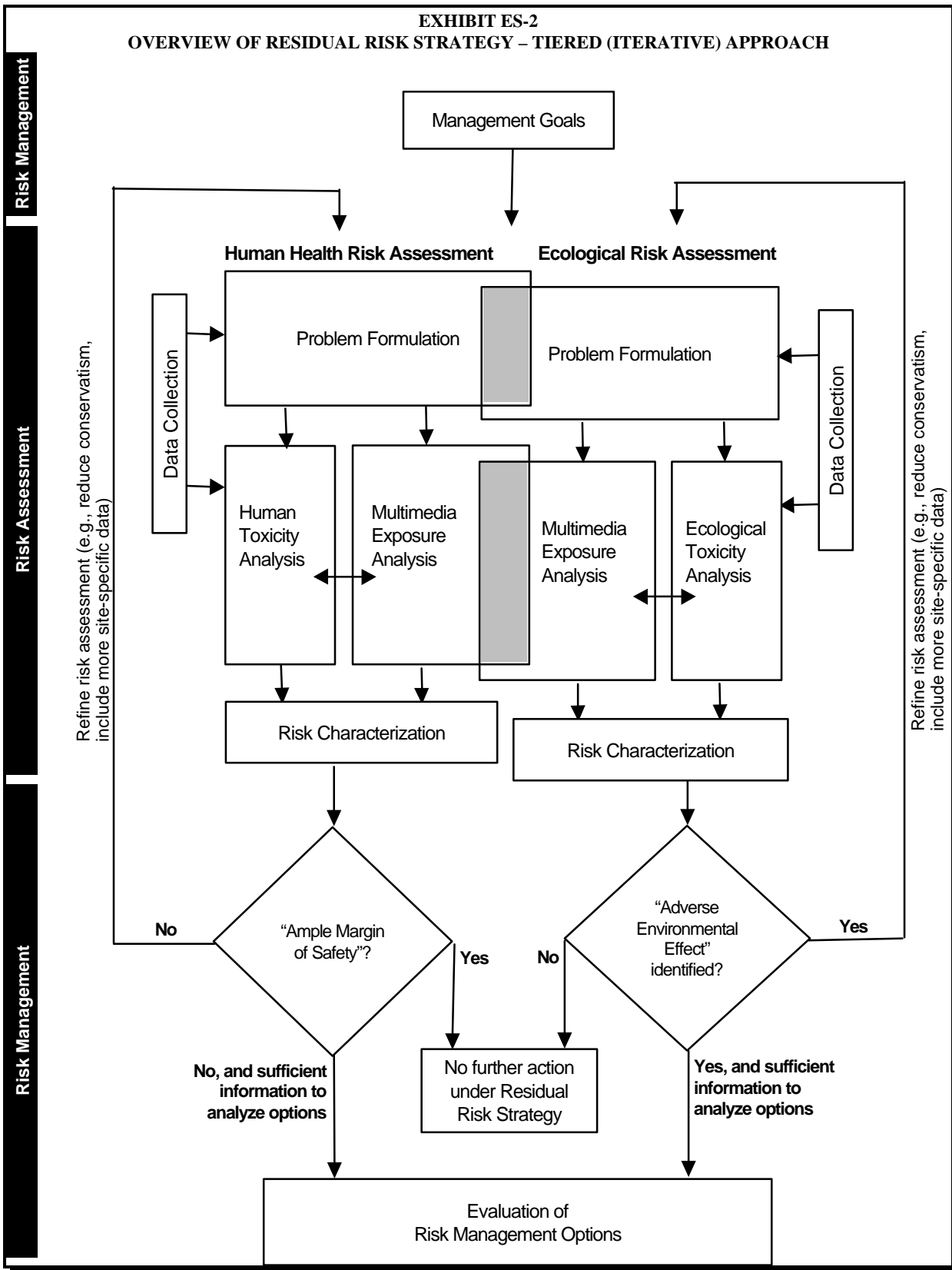
This chapter discusses the residual risk provisions of CAA sections 112(f)(2) through (6), describes EPA's overall residual risk strategy, and presents an ecological risk decision framework for HAPs. EPA has developed the residual risk strategy to implement the requirements of CAA sections 112(f)(2) through (6). Those sections require EPA to promulgate standards beyond MACT when necessary to provide "an ample margin of safety to protect public health" and to "prevent, considering costs, energy, safety, and other relevant factors, an adverse environmental effect." Goals of the residual risk strategy include: (1) assessing any risks remaining after MACT standard compliance; (2) determining if additional emission reductions are necessary and, if so, for which source categories; (3) setting a standard that protects the public with an "ample margin of safety;" and (4) setting a more stringent standard, if necessary, to protect the environment. EPA's intent is to implement a residual risk strategy that will allow the Agency to be flexible in its decisions while ensuring that public health and the environment are protected. EPA's objectives also include continuing the partnership with State/local programs in the sharing of data and expertise, and including all groups who may be affected by residual risk decisions (e.g., industry, public interest groups) as part of the process.

**Exhibit ES-2** is a flowchart representing the general residual risk strategy. In short, the strategy calls for an iterative, tiered assessment of the risks to humans and ecological receptors through both direct and multipathway exposures to HAPs. The first component of the residual risk strategy is that EPA state its management goals, which are identified in the CAA legislation as described in the paragraph above. EPA may decide to translate those legislative objectives into more specific management goals. Those management goals help direct the problem formulation phase of both the human health and ecological risk assessments. Both assessments use an iterative, tiered approach. The basic premise of the tiered approach is that early tiers are generally screening in nature, which means that they are designed to be relatively simple, inexpensive, and quick, using existing data, defined decision criteria, and models with simplifying assumptions as

inputs. Later tiers refine some or many aspects of the analysis, depending on which can influence risk most and are most uncertain. Later tiers require more effort, but produce results that are less uncertain and less conservative (i.e., less likely to overestimate risk).

As shown in Exhibit ES-2, each tier of the human health and ecological risk assessments is organized into three phases: (1) the problem formulation phase, in which the context and scope of the assessments are specified; (2) the analysis phase, in which the HAPs' toxicity and exposure to humans or ecological receptors are evaluated; and (3) the risk characterization phase, in which the toxicity and exposure analyses are integrated to determine whether a risk exists. As illustrated in Exhibit ES-2, the problem formulation and analysis phases of the human health and ecological risk assessments will partially "overlap" in that, certain pathways of concern for humans (e.g., consumption of contaminated fish) might be also be pathways of concern for ecological receptors (e.g., fish-eating wildlife). The multimedia exposure analyses for both assessments also will overlap because some of the same data and exposure models will be used in both assessments.

The screening risk assessment results will be used to eliminate consideration of low-risk source categories, prioritize the remaining source categories for refined assessments, and also focus the refined assessments so that they can be done more efficiently. While the screening analyses can serve as a basis for a decision to pursue additional analyses or to eliminate low-risk source categories from further consideration under section 112(f), they are not adequate to serve as a basis for establishing additional emission reduction requirements. The information provided by the more refined assessments ultimately will be used by EPA to make decisions on whether additional emission reductions are needed for individual source categories. EPA plans to set priorities for analysis of the 174 source categories based on a number of considerations, including the actual MACT promulgation dates for source categories (which starts the clock on the statutory eight-year time period for residual risk determinations) and any available information bearing on the level of residual risks attributable to various source categories.



## 1. Introduction

In 1990, Congress amended section 112 of the Clean Air Act (CAA) and mandated a new approach to the regulation of hazardous air pollutants (HAPs).<sup>1</sup> Under the original CAA (1970), air toxics were addressed through a risk-based program, and emission standards were set for individual pollutants. The new approach first requires the development of technology-based emission standards for major and area sources of 188 hazardous air pollutants under section 112(d). The statute directs that these standards are to be developed over a 10-year time frame and based on the maximum achievable control technology (MACT). The EPA is currently in the process of developing MACT standards for 174 categories of HAP sources, with completion scheduled in the year 2000. As of September 1997, MACT standards had been promulgated for 48 source categories, resulting in estimated HAP reductions when fully implemented of approximately 980,000 tons per year plus additional significant reductions of particulates and volatile organic compounds (EPA 1997j).

### SECTION 112(f)(1) REPORT REQUIREMENTS

"... the Administrator shall investigate and report, after consultation with the Surgeon General and after opportunity for public comment, to Congress on:

- ▶ Methods of calculating the risk to public health remaining, or likely to remain, from sources subject to regulation under this section after the application of standards under subsection (d) of this section;
- ▶ The public health significance of such estimated remaining risk and the technologically and commercially available methods and costs of reducing such risks;
- ▶ The actual health effects with respect to persons living in the vicinity of sources, any available epidemiological or other health studies, risks presented by background concentrations of hazardous air pollutants, any uncertainties in risk assessment methodology or other health assessment technique, and any negative health or environmental consequences to the community of efforts to reduce such risks; and
- ▶ Recommendations as to legislation regarding such remaining risk."

Section 112(f) of the CAA, in addition to requiring the present Report to Congress (Report), calls for an evaluation of the health and environmental risks remaining after technology-based standards have been promulgated (i.e., **residual risks**) and requires more stringent regulation if certain criteria are not met. Specifically, its focus is to achieve a level of protection that protects the public health with an "ample margin of safety" (see Section 2.1 for a discussion of this term) while also ensuring that residual emissions do not result in "an adverse environmental effect" (defined in CAA section 112(a)(7); see Section 2.2.2). The accompanying text box outlines the requirements in section 112(f)(1) that this Report addresses.

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<sup>1</sup> The Clean Air Act defines hazardous air pollutant as any air pollutant listed under section 112(b), and also provides procedures for adding and deleting pollutants from the list. The terms "**hazardous air pollutants**," "**HAPs**," and "**air toxics**" are used throughout this report synonymously to refer to the pollutants listed under section 112(b).

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## **1.1 Scope of Report**

This Report responds to the statutory directives in section 112(f) of the CAA and also provides EPA's strategy for assessing residual risk remaining from the HAPs being emitted from source categories subject to MACT standards. Chapter 2 provides a brief legislative and regulatory background on the CAA air toxics program in order to provide context for what follows. Chapter 2 also provides a short history of the development of risk-based programs and of risk assessment as the primary tool used by EPA to analyze the potential impacts of air toxics emissions on the exposed population and environment. As discussed in Section 2.3, the development of EPA's risk-based program for air toxics has incorporated input from the National Research Council, the Commission on Risk Assessment and Risk Management, State and local air toxics programs, and a variety of risk assessment policies and guidelines developed (and in some cases under development) by EPA. The Report then addresses, in Chapters 3 and 4, the required statutory elements, as shown in the text box on page 1. Chapter 3 provides information on the methods for conducting human and ecological risk assessments for emissions of air toxics, describes the data required, and the methods for evaluating mixtures. Chapter 4 addresses the remaining statutory elements listed in CAA sections 112(f)(1)(B), (C), and (D) in the order listed in the CAA. In Chapter 5, the Report describes EPA's strategy to conduct residual risk analyses as well as discusses other provisions in section 112(f)(2) through (6) of the CAA. Appendix A provides the full text of CAA section 112(f), Appendix B provides relevant text from the preamble to the 1989 national emission standard for benzene, and Appendix C presents the schedule for promulgation of MACT standards for industry source categories.

The intent of this Report is to address the legislative requirements of section 112(f)(1) and to provide the reader with a basic understanding of how EPA will conduct its risk analyses and make decisions concerning these risk assessments. The methodology descriptions provided are not presented as strict guidance but are discussed with enough detail to inform the reader of EPA's intentions and directions in implementing the "residual risk" analyses. The EPA prefers to be flexible in this process so that as changes are made in the way risk assessments are conducted, they may be incorporated as needed. This flexibility is important because the residual risk program has a potential life span equal to that of MACT, i.e., 10 years.

It is important to note that this Report does not contain the results of any residual risk analyses or information on EPA's potential actions after conducting such analyses (e.g., additional emission reductions for a given source category). The EPA is currently collecting existing data on source categories for which MACT standards have been promulgated and will begin analyzing these data using the proposed strategy.

Congress also requested that EPA report on additional elements related to residual risk, such as the public health significance of such risks and the consideration of background concentrations of toxics. Without having any actual residual risk analyses, it is not possible to draw conclusions about these elements. This Report, however, presents these elements as they apply to residual risk assessments.

Public Health Significance. The EPA is proposing to use the “ample margin of safety” concept, discussed in Section 2.1 of this Report, as the basis for determining the significance of any residual risks for individual source categories. Risk that is judged to be significant under this framework would be subject to regulation. As residual risk assessments are completed for individual source categories, EPA will evaluate public health significance as part of its decision-making process.

In making its regulatory decisions for air toxics, EPA has emphasized carcinogenicity as an endpoint. However, not all air toxics are carcinogens nor do they only affect human health. The EPA acknowledges its mandate to address non-cancer and ecological effects under the residual risk program, while noting that currently there are no policies in place for considering them in air management decisions. The EPA is developing guidance for making these risk management decisions, but they will not be developed in time to be incorporated into this Report.

Technologically and Commercially Available Methods and Costs. This Report describes a range of control options if it is determined that additional control is needed. The Report provides an overview of these options, with an emphasis on pollution prevention approaches.

Actual Health Effects Information. The information available on actual health effects resulting from exposure to air toxics is limited. This Report presents a summary discussion of epidemiological data, laboratory data, and other exposure study data. It also briefly describes how EPA intends to use these data and any actual source category-specific health effects data that may become available when residual risk assessments are conducted.

Background Concentrations. This Report discusses general information on background levels of HAPs, including EPA’s cumulative risk policy in development, and presents a definition of background concentrations for air toxics and residual risk purposes. It describes approaches used by other EPA programs and includes examples of rules and guidance that consider the issue of background. It also presents a discussion of the difficulties in addressing background concentrations in residual risk analyses and identifies data needs to assess background. The discussion concludes by describing EPA’s options to analyze and consider background concentrations in residual risk analyses.

Negative Health or Environmental Consequences to Communities. Congress required EPA to consider negative health or environmental consequences to communities from efforts to reduce residual risks. The EPA interprets this requirement to mean that any risk management options for reducing residual risks must consider other possible health consequences to the community resulting from those decisions. The EPA is aware that pollution control technologies targeted at a single pollutant (e.g., a specific HAP) and single medium (e.g., air), especially conventional end-of-the-pipe treatment technologies,

can inadvertently transfer pollutants and risks to different media, different locations, and different receptors, and can unintentionally create new and different risks in the process of controlling the targeted risk. Thus, EPA intends, as it conducts its residual risk analyses and any subsequent standard-setting actions, to identify potential negative health and environmental consequences when possible and consider the risk-risk tradeoffs associated with any standards established under the residual risk program.

Legislative Recommendations. Congress required the EPA to make “legislative recommendations regarding any identified residual risk.” The EPA has interpreted this Congressional requirement to mean that if an unacceptable residual risk were identified, and no current authority within the CAA were determined to be adequate to reduce that risk, then EPA would propose an approach that would assure that risk reductions would occur. The EPA believes that the regulatory approach embodied in the CAA is adequate for maintaining the goal of protecting the public and environment’s health, and, therefore, is not proposing any legislative changes.

## **1.2 Peer Review**

The EPA is fully committed to environmental protection that is founded on sound and credible science. Objective, independent peer review of the scientific and technical bases of the Agency’s actions is critical to accomplishing the Agency’s mission. The Agency’s commitment to credible, effective peer review is stated in its Peer Review Policy of June 7, 1994. Full implementation of this policy remains an Agency priority.

Most of the major references that form the foundation of this Report to Congress have undergone (or are currently undergoing) external peer review. In addition, EPA intends to have this Report peer reviewed during the public comment period because it outlines specific applications for the methods and policies contained in these references. For example, EPA believes that it is necessary to obtain an independent evaluation of questions such as whether the Report identifies the most relevant and useful methods of assessing risks from stationary sources and whether it properly characterizes the types of data on which these methods rely. The results of this peer review will be incorporated into the final Report.

## **2. Background: CAA Air Toxics Program and the Development of Risk Assessment Methods**

In order to understand the mandate of CAA section 112(f) and the purpose behind its charge to EPA, it is helpful to understand the legislative approach used to regulate HAPs in the 1970 CAA Amendments, the subsequent regulatory history in the 1970s and 1980s, and the legislative strategy behind the approach taken by the 1990 CAA Amendments. It is also useful as background to consider some of the history and key events in the development of risk assessment methods and policy for EPA's air toxics program.

### **2.1 Legislative Approach and Regulatory History of the Air Toxics Program: 1970-1990**

Congress first required regulations limiting emissions of HAPs in 1970 by including an air toxics provision in the 1970 CAA Amendments. This provision described a health-based program that required EPA to identify and list HAPs based on human health criteria described in the Amendments. The EPA was to then promulgate standards for each pollutant, on a source category-by-source category basis, at a level that would ensure the protection of public health with "an ample margin of safety." After EPA listed a pollutant, regulation was required within a short time.

The EPA did not produce many air toxics regulations under the program established by the 1970 CAA Amendments. In the 20 years following the enactment of this legislation, EPA identified eight pollutants as HAPs and regulated seven of these. Impediments to regulation included the amount and type of data needed to establish a chemical as a HAP; emissions standards based on what the Agency interpreted to be solely human health effects considerations; extremely short statutory deadlines; and disagreements over how health effects should be assessed. A common theme running through many of these impediments to regulatory action was the lack of a consistent risk management framework with which to make regulatory decisions.

The most significant example of EPA's attempts to regulate HAPs under the 1970 CAA Amendments resulted in a DC Circuit Court decision that would guide the development of EPA's risk management approach for air toxics (*Natural Resources Defense Council (NRDC) v. EPA* 1987). NRDC sued EPA on the Agency's attempt to establish a national emission standard (NESHAP) for vinyl chloride, stating that the Agency improperly used cost in regulating this HAP. The U.S. Court of Appeals for the D.C. Circuit court agreed with NRDC, and in its decision presented a two-step framework by which to apply the "ample margin of safety" language: (1) first determine a "safe" or "acceptable risk" level considering only public health factors, and (2) then set an emission standard that provides an "ample margin of safety" to protect the public health, considering relevant factors in addition to health such as costs, economic impacts, technical feasibility, uncertainties, and any other relevant factors.

The 1989 NESHAP for benzene (EPA 1989b) presented the following risk management framework for cancer risk, which reflects the two-step approach suggested by the court. The benzene rule preamble states that in determining acceptable risk:

The administrator believes that an MIR [maximum individual risk] of approximately 1 in 10 thousand should ordinarily be the upper end of the range of acceptability. As risks increase above this benchmark, they become presumptively less acceptable under section 112, and would be weighed with the other health risk measures and information in making an overall judgment on acceptability. Or, the Agency may find, in a particular case, that a risk that includes MIR less than the presumptively acceptable level is unacceptable in light of the other health risk factors (EPA 1989b, p. 38045).

The EPA believes that the level of the MIR, the distribution of risks in the exposed population, incidence, the science policy assumptions and uncertainties associated with risk measures, and the weight of evidence that a pollutant is harmful to health are all important factors to be considered in the acceptability judgment (EPA 1989b, p. 38046).

The preamble also states that in the second step, where the standard is set with an ample margin of safety:

EPA strives to provide protection to the greatest number of persons possible to an individual lifetime risk level no higher than approximately 1 in 1 million. In the ample margin decision, the Agency again considers all of the health risk and other health information considered in the first step. Beyond that information, additional factors relating to the appropriate level of control will also be considered, including costs and economic impacts of controls, technological feasibility, uncertainties, and any other relevant factors (EPA 1989b, p. 38046).

Thus, the benzene NESHAP established specific risk management policy for cancer risks, including some numerical criteria. It did not, however, explicitly address non-cancer human health effects or environmental effects. Appendix B provides excerpts of the preamble text from the 1989 benzene NESHAP.

The HAP provisions of the 1970 CAA Amendments were written specifically in terms of public health effects, with no mention of ecological or environmental effects anywhere in section 112. In its original form, CAA section 112(b) directed that NESHAPs be set to provide "...an ample margin of safety to protect the public health..." In fact, HAPs were defined specifically in terms of human health; section 112(a) of the 1970 CAA defined a HAP as an air pollutant that "...may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." Thus, there was no legislative directive to consider environmental effects in regulating HAPs in the pre-1990 air toxics program.

## **2.2 Legislative Strategy For Air Toxics: Post-1990**

### **2.2.1 Changing the Regulatory Approach**

Recognizing that the "health test" (i.e., the requirement for the protection of public health with an "ample margin of safety") was the most contentious part of section 112 under the 1970 CAA Amendments, Congress shifted the focus from individual pollutants to industrial source categories and developed a phased approach to controlling air toxic emissions in the 1990 CAA Amendments. Congress initially listed 189 HAPs in section 112(b), one of which has since been delisted by EPA. As part of the first phase of the new air toxics program, EPA must promulgate national, technology-based emission standards for sources in 174 source categories emitting any of the 188 listed HAPs above specific emission thresholds. The overall approach is to use available control technologies or work practice changes to get emission reductions in a timely manner for as many of the listed HAPs as possible, regardless of a HAP's inherent toxicity and potential risk. This technology-based standards program is commonly referred to as the MACT (Maximum Achievable Control Technology) program. Although there is no health test in this phase, it is intended that effective MACT standards will reduce a majority of the HAP emissions and much of the significant risk.

As part of the second phase of the program, EPA is to conduct specific studies to assess the potential for adverse effects and, if necessary, take action to reduce the potential for these effects. These studies include the Mercury Report to Congress (EPA 1997k), the Great Waters Study (EPA 1997l), and the Utilities Study (EPA 1998). The Agency is also required to create a program to address air toxics in urban areas, and to develop a program to assess post-MACT residual risks and set health based-standards if necessary to protect the public health, or more stringent standards to protect against adverse environmental effects.

The revised air toxics legislative strategy embodied in the 1990 Amendments maintains the goal of protecting the public health and the environment and provides a more complete strategy for dealing with a variety of adverse effects. The strategy recognizes that not all problems are national in scope or have a single solution. National emission standards must be promulgated to decrease the emissions of as many HAPs as possible from stationary sources and some area sources, but authority is also provided to look at smaller scale problems such as the urban environment or the deposition of HAPs to large water bodies in order to address specific concerns. The strategy also recognizes the need to focus or prioritize efforts to meet specific needs such as a concern for a class of toxic and persistent HAPs. There are mechanisms for increasing partnerships among EPA, States, and local programs in order to address problems specific to these regional and local environments.

In summary, Congress developed a strategy that, when taken as a whole, provides EPA with the flexibility to address a wide range of air toxics problems. The provisions of this strategy describe the approaches for identifying the nature and scope of the problem and the mechanisms for involving all concerned parties in discussions. Congress' strategy provides a diversity of authorities for managing the identified risk in a cost-effective way while protecting human and environmental health in the process.

## **2.2.2 The Residual Risk Program**

To ensure that MACT regulations protect public health and the environment, Congress included section 112(f) in the 1990 CAA Amendments, which requires a human health risk- and adverse environmental effects-based “needs test” in the second regulatory phase of the air toxics program (see Appendix A for full text of section 112(f)). In this phase, referred to as residual risk standard setting, EPA is required to promulgate additional standards for those source categories that, after imposition of MACT standards, are emitting HAPs at levels that present a potential unacceptable risk to the public or the environment. Congress directed that such residual risk standards should “provide an ample margin of safety to protect public health.”

Section 112(f) also specifically gives EPA the mandate to consider environmental health in its assessment. Although not very explicit as to how this should be done, Congress does say that EPA shall promulgate standards to provide an ample margin of safety to protect public health unless the Administrator determines that a more stringent standard is necessary to prevent “an adverse environmental effect.” The statute directs that consideration of adverse environmental effects must take into account “costs, energy, safety, and other relevant factors” in deciding what level is protective. Adverse environmental effect is defined in section 112(a)(7) as “any significant and widespread adverse effect, which may reasonably be anticipated, to wildlife, aquatic life, or other natural resources, including adverse impacts on populations of endangered or threatened species or significant degradation of environmental quality over broad areas.”

## **2.3 Development of Human Health and Ecological Risk Assessment Methods**

This section describes some of the history and key events leading up to development of EPA’s general air toxics risk assessment methodology, and to development of the specific residual risk strategy described in this Report. Identifying the nature and scope of the various air toxics problems through data collection, analysis, and mandated studies is a first step in implementing the post-1990 strategy. Risk assessment is the primary method to be used in determining the magnitude of potential impacts resulting from continued HAP exposures – that is, the residual risks. Congress included mechanisms that would assist in the development of the residual risk assessment process, including the reports discussed in the next two sections. EPA has built on its existing (and continuously evolving) risk assessment policies and guidance, and also has taken into account State and local air toxics risk programs.

### **2.3.1 National Academy of Sciences Reports of 1983 and 1994**

The NAS has on several occasions been requested by Congress to evaluate and discuss the processes of risk assessment and risk management. Two of their studies, published in 1983 and 1994, are especially relevant as a foundation for this Report. The emerging practice of risk assessment at EPA and other Federal agencies spurred Congress to commission a report from the

National Research Council (NRC) of the NAS in the early 1980s. The result was the landmark 1983 study entitled *Risk Assessment in the Federal Government: Managing the Process* (NRC 1983). This report was written at a time when there was an increasing concern about the risk of cancer resulting from exposure to chemicals in the environment — the fear was that policy might not keep up with the state of the science, which was changing very rapidly in this area.

#### **PURPOSE OF THE 1983 NRC REPORT**

The 1983 NRC report was intended to:

- ▶ "Explore the intricate relations between science and policy" in the field of risk assessment; and
- ▶ "Search for the institutional mechanism that best fosters a constructive partnership between science and government."

The 1983 NRC report recognized the importance of the relationships that exist between science and risk assessment, and between risk assessment and risk management, and undertook the task of clearly defining these relationships. The NRC acknowledged that risk assessment must take full advantage of the available science while maintaining the need to accommodate the various regulatory requirements, and that risk assessment was only one component of the risk management decision process. To define this more clearly, the NRC made a series of recommendations. In general, the NRC recommended the development of specific guidelines for performing risk assessments (at that time, cancer was the main endpoint of concern), that risk assessments developed using the guidelines be reviewed and distributed to the public, and that these risk assessments clearly distinguish the science and policy components from the political, economic, and technical considerations that influence the risk management decisions. This report also provided a description of the health risk assessment paradigm that continues to serve as EPA's model. Partly in response to this report, EPA began a process that continues today of publishing Agency-wide guidelines addressing important areas of risk assessment (see Sections 2.3.3 and 2.3.4).

#### **STEPS INTEGRAL TO ANY RISK ASSESSMENT**

The NRC risk assessment paradigm includes four steps that are integral to any risk assessment (NRC 1983, NRC 1994):

- ▶ Hazard identification
- ▶ Dose-response assessment
- ▶ Exposure assessment
- ▶ Risk characterization

The NRC's follow-up report, *Science and Judgment in Risk Assessment* (NRC 1994), mandated by Congress under section 112(o) of the CAA, took a closer look at current risk assessment methods, with a statutorily directed focus on carcinogenic risk. The intent (and mandate) of the report was not to look at EPA's regulatory decisions but the methods used to support those decisions. The NRC committee observed that several themes were common to all elements of the risk assessment process and noted that these themes were usually the focal points for criticisms of specific risk assessments. The themes discussed included the use of default assumptions; the available data; uncertainty and variability; assessment of multiple chemical exposures, multiple routes of exposure, and the potential for multiple adverse effects; and steps taken to validate the methodologies used throughout the risk assessment process. NRC's concerns, discussions, and recommendations were viewed as a way to increase the effectiveness

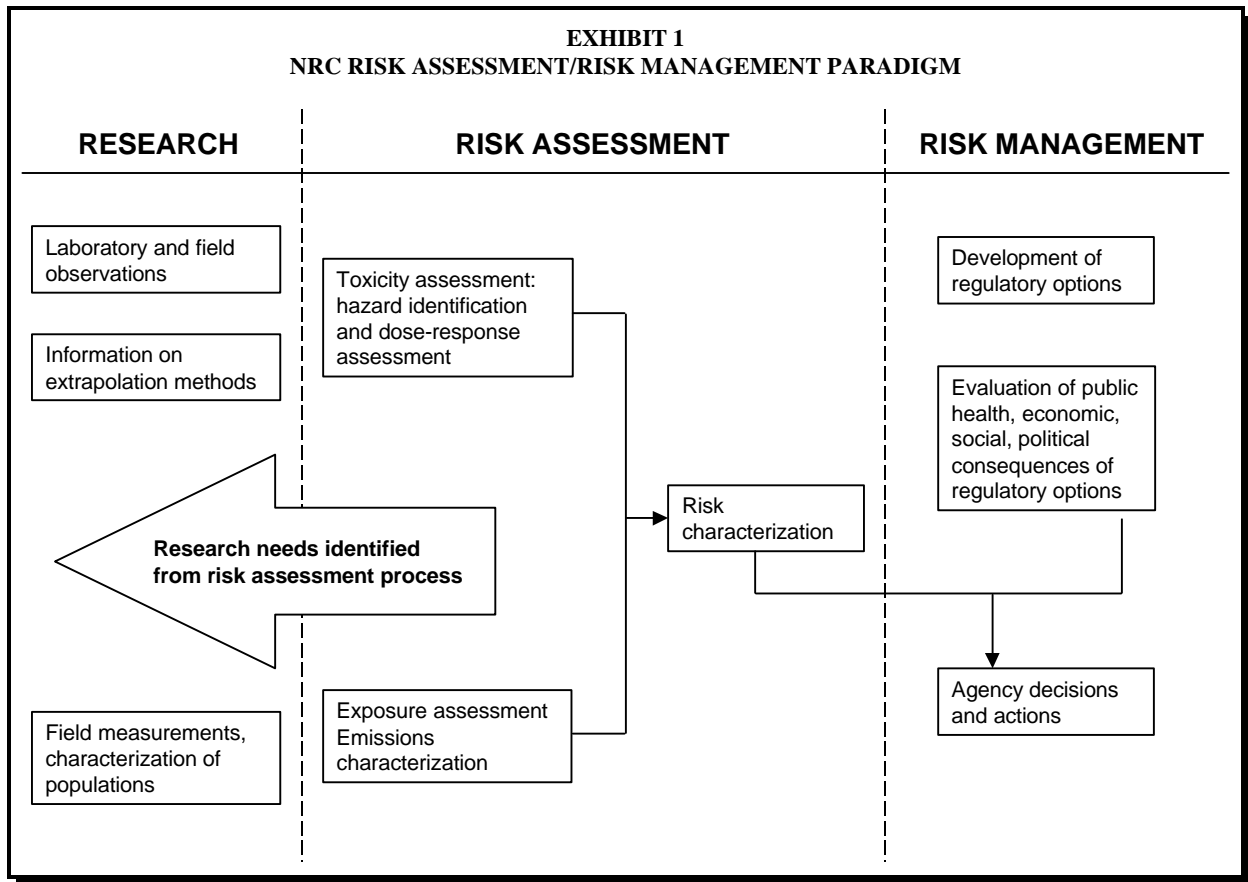
and accuracy of the risk process defined in their 1983 report. **Exhibit 1** shows the risk assessment/risk management paradigm as presented in the 1994 NRC report.

The NRC concluded that “because of limitations on time, resources, scientific knowledge, and available data, EPA should generally retain its conservative, default-based approach to risk assessment for screening analysis in standard-setting; however, several corrective actions are needed to make the approach more effective.” The NRC went on to say:

- EPA should continue to regard the use of default options as a reasonable way to deal with uncertainty about underlying mechanisms in selecting methods and models for use in risk assessment;
- EPA should explicitly identify each use of a default option in risk assessment;
- EPA should clearly state the scientific and policy basis for each default option; and
- The Agency should consider attempting to give greater formality to its criteria for a departure from default options, in order to give greater guidance to the public and to lessen the possibility of *ad hoc*, undocumented departures from default options that would undercut the scientific credibility of the Agency’s risk assessment process. At the same time, the Agency should be aware of the undesirability of having its guidelines evolve into inflexible rules.

The committee recommended that EPA develop and use an iterative approach to health risk assessments to delist source categories and eliminate residual risk. The NRC also proposed a possible iterative approach that will allow for improvements in the default-based approach by improving both models and the data used in analysis. Furthermore, the committee suggested that EPA present not only point estimates of risk, but also the sources and magnitudes of uncertainty associated with these estimates.

The NRC also discussed how the risk assessment recommendations in its report could be implemented in the context of section 112. Section 112 calls for EPA to regulate HAPs in two stages. In the first, sources would be required to do what is feasible to reduce emissions based on currently available technology. In the second, EPA would implement the proposed iterative approach. Using this approach, EPA would set residual risk standards to protect public health with an ample margin of safety if it concluded that implementation of the first stage of standards did not provide such a margin of safety. The committee indicated that neither the resources nor the scientific data exist to perform a full-scale risk assessment on all the chemicals listed as HAPs and their sources. Therefore, the committee supported an iterative approach to risk assessment of HAPs. This approach would start with relatively inexpensive screening techniques and move to more resource-intensive levels of data-gathering, model construction, and model application as the particular situation warranted. The result would be a process that supports the risk management decisions required by the CAA and that provides incentives for further research, without the need for costly case-by-case evaluations of individual chemicals at every facility in every source category. It also recommended a priority-setting scheme based on initial assessments of each chemical’s possible impact on human health and welfare. In many ways,



EPA has been headed in the directions recommended by this report, and it continues to do so as it moves into the risk-based phase of the CAA legislative strategy for HAPs.

### 2.3.2 Commission on Risk Assessment and Risk Management

Section 303 of the 1990 CAA Amendments mandated formation of the Commission on Risk Assessment and Risk Management (CRARM) in response to unresolved questions about the approach EPA should take to determining whether any significant risks to human health remain after the implementation of the CAA Amendments' technology-based emission controls. On June 13, 1996, the CRARM released a draft of its report, *Risk Assessment and Risk Management in Regulatory Decision-Making* (CRARM 1996). At the completion of the public comment period, the CRARM announced that it planned to release its final report in two parts. Volume I, released in January 1997, focuses on the framework for environmental health risk management (CRARM 1997a). Volume II, released in March 1997, addresses a variety of technical issues related to risk assessment and risk management, including margin of exposure, management of residual risks from air toxics, comparative risk, decision criteria, uncertainty analysis, and recommendations to specific agencies (CRARM 1997b).

The CRARM's framework fosters an integrated approach to addressing complex, real-world issues that affect more than one environmental medium and involve exposures to mixtures

of chemicals. The CRARM anticipates that its framework will assist Congressional committees and subcommittees, and government agencies (e.g., EPA, DOE), in developing integrated approaches to environmental risk management.

### **The Commission's Mandate**

The Commission's mandate was to investigate "the policy implications and appropriate uses of risk assessment and risk management in regulatory programs under various Federal laws to prevent cancer and other chronic health effects which may result from exposure to hazardous substances" (CRARM 1996, 1997a, and 1997b). The CRARM's final report indicated that the Commission's mandate included:

- Assessing uses and limitations of risk assessment and economic analysis in regulatory decision-making (e.g., setting emission, ambient, and exposure standards for hazardous substances);
- Considering the most appropriate methods for measuring and describing cancer risks and non-cancer chronic health effects risks from exposures to hazardous substances;
- Evaluating exposure scenarios for risk characterization (e.g., use of site-specific exposure data in setting emissions standards);
- Determining how to describe and explain uncertainties (e.g., associated with measurement, extrapolation from animal data to humans);
- Discussing approaches to determining the existence of synergistic or antagonistic effects of hazardous substances;
- Enhancing strategies for risk-based management decisions;
- Considering the desirability of developing a consistent standard of acceptable risk across various Federal programs;
- Suggesting ways to improve risk management and risk communication;
- Commenting on the conclusions in the NRC report *Science and Judgment in Risk Assessment*; and
- Making recommendations about peer review.

Although the Commission's mandate was limited to "cancer and other chronic human health effects," the group did discuss ecological risk assessment for the following reasons:

- Human health is related to the health of the environment;
- Principles of health risk assessment are relevant to ecological risk assessment; and
- Economic analyses should not be limited to human health benefits.

### **Recommendations from the CRARM Final Report**

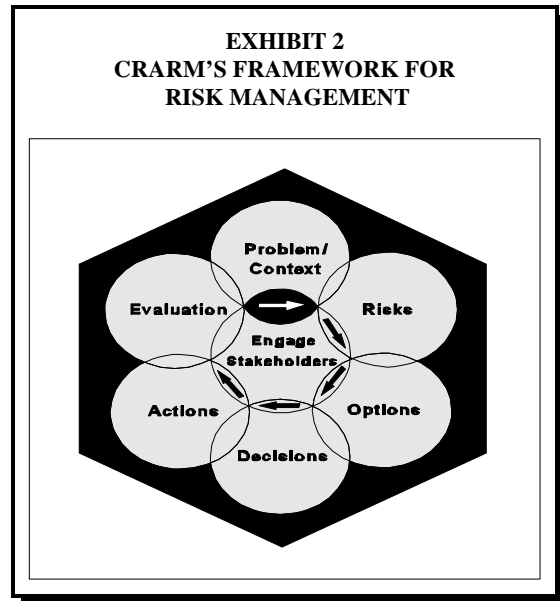
The CRARM final report presents several recommendations relevant to risk management and residual risk in the context of air toxics. These recommendations focus on the following topic areas: (1) risk management framework; (2) comparative risk assessment; (3) margin of exposure; (4) margin of protection; (5) realistic exposure scenarios; (6) cost benefit analysis; (7) interagency consistency; and (8) residual risk scheme.

**Risk Management Framework.** The Commission's proposed framework for environmental health risk management is presented graphically in **Exhibit 2**. The framework calls for some level of stakeholder involvement during each of the six stages of risk management. In fact, stakeholder collaboration is the central element in the framework. In addition, the framework is designed to be iterative. If appropriate, the risk problem can be redefined and reassessed as new data and new views are found.

Another key principle of the framework is that risk management should explicitly consider the comprehensive real-world context of a risk problem, rather than limit the problem's context to one that considers only one type of risk associated with a single chemical in a single environmental medium. The Commission identified several risk management contexts:

- Multisource context (e.g., the population may be exposed to the same pollutant from sources other than the one in question);
- Multimedia context (e.g., exposure to the pollutant may be occurring from other environmental media);
- Multichemical context (e.g., other pollutants from the same source may pose additional risks); and
- Multirisk context (e.g., the magnitude of risk from one problem may be insignificant compared to similar risks that a population faces from other stressors).

According to the Commission's framework, the relevant contexts for a risk problem are first identified and characterized in the problem/context phase of risk management. These risk contexts are then refined in the risk analysis phase and are addressed in all of the remaining phases of the risk management process.



**Comparative Risk Assessment.** The CRARM report recommends that Federal agencies try a comparative risk analysis approach on an experimental or demonstration basis to seek consensus on priorities for managing environmental risks. The results of such efforts should influence agency resource allocation. The Commission noted that there is wide disagreement on the efficacy of this approach for setting priorities, and that experience shows there is no guarantee that this process will result in consensus among stakeholders, agencies, and funding authorities. However, the Commission also noted that experience shows that the process itself can help to build coalitions that favor priority shifting and shifting resources to identified priorities.

**Margin of Exposure.** The Commission recommended using a margin-of-exposure approach for expressing risks for carcinogens, in addition to the methods historically used to express risks for carcinogens. The EPA defines a margin of exposure ratio as a specified dose derived from a tumor bioassay, epidemiologic study, or biologic marker study, such as the dose associated with a 10 percent response rate, divided by an actual or projected human exposure (EPA 1996b). Lower margins of exposure indicate greater concern. The margin-of-exposure approach is comparable to the methodology EPA uses to estimate non-cancer effects hazard quotients based on the reference dose (RfD) or the reference concentration (RfC). The Commission felt that, because the bulk of the data for carcinogens is often limited to observable dose-response data from bioassays, expressing cancer risk in terms of predicted incidence or numbers of deaths per unit of population implies an “unwarranted” degree of precision. The Commission argued further that the use of the margin of exposure approach will aid in comparative risk assessment, particularly for comparing risks of carcinogens and noncarcinogens.

**Margin of Protection.** The Commission recommended that estimated acceptable daily intakes (ADIs), RfDs, and RfCs be used in risk assessment and risk management. These values are derived using the margin-of-protection or safety factor method, and represent chemical exposure concentrations that would be associated with negligible risk. The negligible risk levels are calculated by identifying a no-observed-adverse-effect level (NOAEL), a lowest-observed-adverse-effect level (LOAEL), a benchmark dose, or other experimentally-derived level and dividing the value by factors that are designed to account for variability and uncertainty. A margin of protection is the product of the factors (e.g., a margin of protection is 1,000 when three factors of 10 are multiplied together). The Commission pointed out that Europe, Canada, and many other countries use the margin-of-protection approach for carcinogens and non-carcinogens, while the U.S. uses this approach primarily for non-carcinogens.

**Realistic Exposure Scenarios.** The report states that risk management decisions should be based on realistic exposure scenarios, rather than on the maximum exposed individual (MEI), and supports agencies’ recent progress toward this end. The Commission believes that, where possible, exposure assessments should include information about specific groups: infants, children, pregnant women, low-income groups, and minority group communities with exposures influenced by social or cultural practices.

**Cost-benefit Analysis.** The Commission supports the use of economic analysis as a consideration in risk management decisions, but not as the overriding factor in a decision. The report calls for explicit descriptions of assumptions, data sources, sources of uncertainty, and costs across society to be presented in parallel with descriptions associated with risk assessments.

**Interagency Consistency.** In conducting risk assessments, agencies should coordinate their risk assessment methods and assumptions unless there is a specific statutory requirement for different choices. Scientific disagreements should be explained.

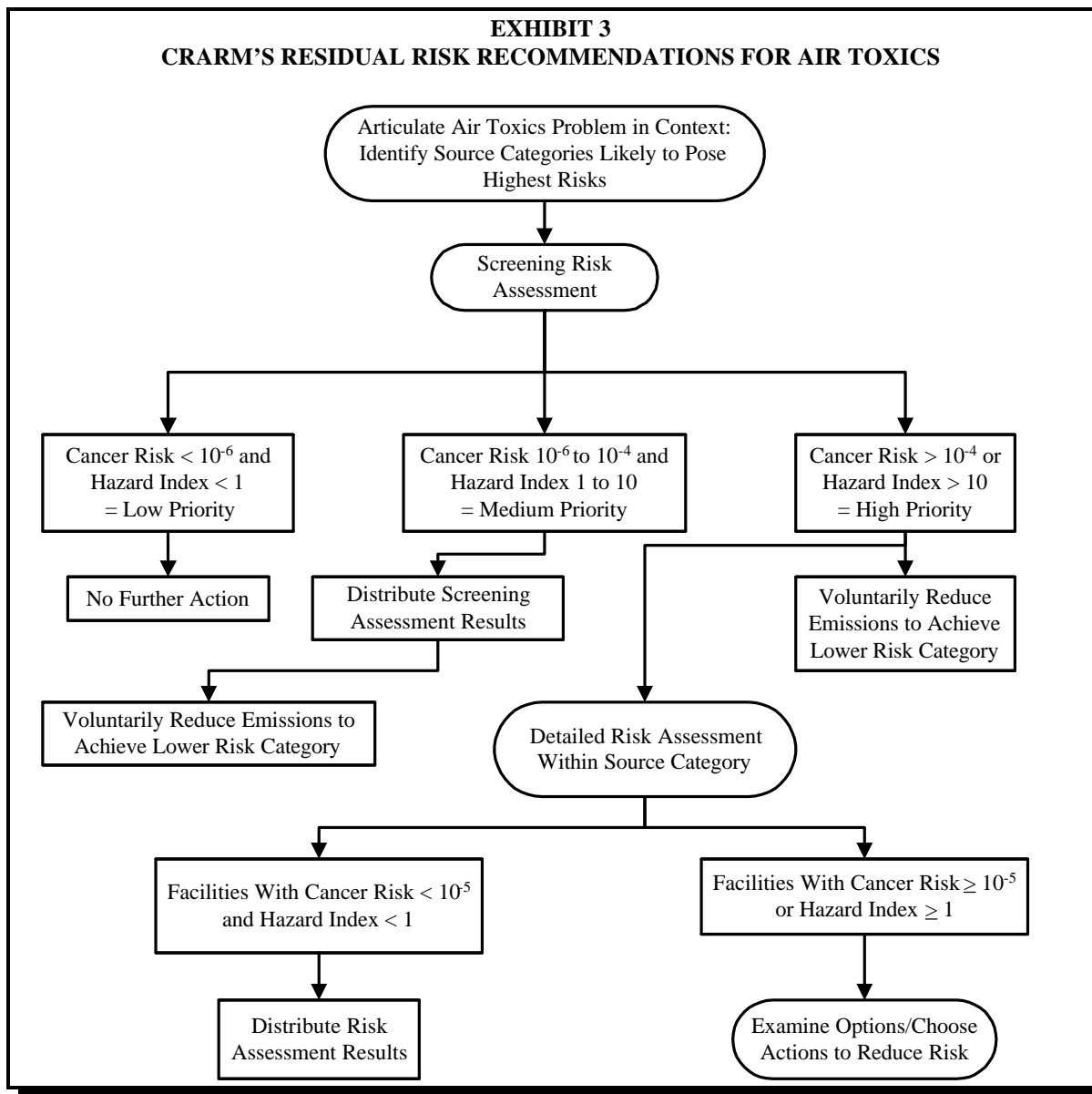
**Residual Risk Recommendations.** The Commission recommended a tiered approach, which is summarized in **Exhibit 3**, to manage residual risks of section 112 CAA HAPs after implementation of the CAA's technology-based (MACT) standards. EPA's approach is similar to the Commission's proposal in that it incorporates both screening-level and refined risk assessments as a basis for priority-setting and decision-making. Specifically, CRARM proposed that EPA develop their approach in accordance with the five recommendations listed in the accompanying text box. A comparison of EPA's strategy with the Commission's recommendations is presented in Section 5.3.5.

**CRARM RECOMMENDATIONS FOR  
RESIDUAL RISK STRATEGY**

- ▶ Characterize and articulate the scope of the national, regional, and local air toxics problems and their public health and environmental contexts.
- ▶ Use available data and default assumptions to perform screening level risk assessments to identify sources with the highest apparent risks.
- ▶ Conduct more detailed assessments of sources and facilities with the highest risks, providing guidance and incentives to regulated parties to either conduct these risk assessments or reduce emissions to below screening thresholds.
- ▶ At facilities that have incremental lifetime upper-bound cancer risks greater than one in 100,000 persons exposed or that have exposure concentrations greater than reference standards, examine and choose risk reduction options in light of total facility risks and public health context.
- ▶ Consider reduction of residual risks from source categories of lesser priority.

**2.3.3 Development of Human  
Health Risk Assessment at EPA**

While the first NRC document on risk assessment in the Federal government was published in 1983, EPA has used risk assessment techniques since its inception in 1970. Some quantitative analysis of cancer and other risk was performed prior to 1970 by the Food and Drug Administration and the Federal Radiation Council. The EPA built on this knowledge soon after its inception by confronting potential hazards associated with pesticide use. After considering available human and non-human toxicity data, EPA restricted domestic use of DDT and other pesticides, in part due to their cancer risks. It was acknowledged by EPA that regulations such as these needed appropriate scientific basis, and thus information on the cancer risks associated with these pesticides was collected through administrative hearings and testimony. Summary documents from these hearings were collectively referred to as the "Cancer Principles." Criticisms of these documents, which were inadvertently perceived as a formal Agency cancer risk assessment policy, led to the development of interim guidelines published by EPA in 1976. Three years later, the Interagency Regulatory Liaison Group (a conglomeration of several federal agencies, including EPA) published additional cancer risk assessment guidelines. At about the same time, cancer risk assessment techniques were used by EPA in the regulation of toxic chemicals covered under the 1976 Toxic Substances Control Act, and by the end of EPA's first decade, risk assessment techniques were being used to develop water quality criteria for potential



carcinogens. Throughout the 1980s, the use of risk assessment in EPA grew significantly and increasingly covered non-cancer risks in addition to cancer risks. During the 1980s, cancer risk assessment techniques were used in the development of national emission standards for air toxics such as vinyl chloride and benzene.

As the use of risk assessment increased in the 1980s, there was a growing awareness of both the lack of standard guidance for and the inconsistencies in the use of risk assessment at EPA. To address this need, the Agency undertook some administrative reforms and published several key guidelines and other policy documents, particularly during the second half of the decade. In response to the 1983 NRC report, the Agency published *Risk Assessment and*

*Management: Framework for Decision Making* (EPA 1984b), designed to address NRC recommendations and help EPA make better and more rapid decisions about environmental toxic chemical problems. Beginning in 1986, EPA has published an influential series of Agency-wide guidelines in the *Federal Register* identifying the best methods for assessing human health risks from environmental pollution. These guidelines (see text box), which cover both cancer and non-cancer risks, are not meant to be static but may be revised as new information and methods become available. EPA's use and development of human health risk assessment has continued to grow through the 1980s and 1990s with the establishment of the Integrated Risk Information System (IRIS) toxicity database, the repository of Agency consensus noncancer RfDs and RfCs, and cancer assessments, studies on comparative risk, and multimedia, site-specific risk

assessments such as those performed through the RCRA and Superfund programs. Since 1995, EPA has published draft risk assessment guidelines on neurotoxicity (EPA 1995b) as well as draft revisions to its carcinogenicity guidelines (EPA 1996b) and mixtures guidelines (EPA 1997g). Human health risk assessment techniques embodied in these guidance documents are the foundation of the estimation of residual risks from air toxics under the CAA.

**EPA HUMAN HEALTH RISK  
ASSESSMENT GUIDELINES**

EPA has published final risk assessment guidelines that address the following areas:

- ▶ Mutagenicity (EPA 1986b)
- ▶ Carcinogenicity (EPA 1986c)
- ▶ Chemical mixtures (EPA 1986d)
- ▶ Developmental toxicity (EPA 1991a)
- ▶ Exposure assessment (EPA 1992c)
- ▶ Risk characterization (1995a)
- ▶ Reproductive toxicity (EPA 1996d)
- ▶ Probabilistic analysis (EPA 1997b)

Draft guidelines have also been issued for neurotoxicity (EPA 1995b), and draft revisions have been issued for carcinogenicity (EPA 1996b) and are under development for mixtures (EPA 1997g).

### **2.3.4 Development of Ecological Risk Assessment at EPA**

The development of ecological risk assessment at EPA began in the 1970s primarily in two program areas, water quality and pesticide registration. The 1972 Clean Water Act (CWA) set objectives for eliminating surface water pollution based on receiving water uses of "fishable, swimmable waters." The 1972 amendments to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) required that pesticides be evaluated for "any unreasonable adverse effects on the environment." Subsequent legislation for environmental protection resulted in the development of other lines of ecological assessment practices in the late 1970s and in the 1980s, each tailored to the mandates of particular statutes (e.g., the Toxic Substances Control Act).

To meet its statutory mandates and promote consistency among assessments within program areas, EPA began developing program-specific guidelines for ecological assessments in the 1980s. To meet its CWA mandate, EPA published standardized guidelines for deriving ambient water quality criteria in 1986 (EPA 1986f). The guidelines specified that the criteria provide a "reasonable amount of protection of most species in an balanced healthy aquatic community" (EPA 1986f). For pesticide registration evaluations, EPA developed a framework for evaluating the effects of pesticides on nontarget organisms such as wildlife or aquatic communities and published these standard evaluation procedures in 1986 (EPA 1986g). Efforts

to develop and document ecological assessment practices in other EPA program offices followed in the late 1980s (e.g., the *Risk Assessment Guidance for Superfund, Volume II: Environmental Evaluation Manual* (EPA 1989d)).

By the mid and late 1980s, EPA recognized a need for consistency in evaluating ecological risks across program offices and a need to make its ecological research efforts more responsive to ecological risk assessment needs Agency-wide. In response, the Office of Research and Development (ORD) began an evaluation of program-specific ecological risk assessment practices and initiated development of guidelines to establish a consistent and scientific basis for assessing ecological risks associated with toxic substances, for use Agency-wide. EPA's Risk Assessment Forum assumed responsibility for the Guidelines in 1990 and initiated three ecological risk guidance projects: (1) a "framework" to describe the basic principles for ecological risk assessment; (2) a set of case studies to illustrate the "state-of-the-practice" in ecological assessments; and (3) a long-range plan for developing specific ecological risk guidelines.

To accommodate the diverse kinds of ecological risk assessments conducted across program offices at EPA, the Agency found it necessary to modify the 1983 NRC paradigm for risk assessment. Most notably, EPA added a problem formulation phase to the beginning of the ecological risk assessment process. In problem formulation, the scope, context, and ecological values of concern are identified. In 1992, EPA published its *Framework for Ecological Risk Assessment* (EPA 1992a). As the foreword of that document states, "use of the framework ... is not a requirement within EPA, nor is it a regulation of any kind. Rather, it is an interim product that is expected to evolve with use and discussion." As an interim method of providing more detailed guidance for its different program offices, EPA published two volumes of *A Review of Ecological Assessment Case Studies from a Risk Assessment Perspective* (EPA 1993d, 1994g). The case studies are wide-ranging in scope, representing a variety of ecosystems, ecological endpoints, chemical and non-chemical stressors, and programmatic requirements within EPA, and illustrate how the *Framework* could be applied in each case.

The Forum's goal was to develop detailed guidelines for ecological risk assessment that would be a counterpart to the existing EPA health risk guidelines. In August 1996, EPA published its *Proposed Guidelines for Ecological Risk Assessment* for public review and comment (EPA 1996c). That document expands on some *Framework* concepts and modifies others to reflect Agency experiences in the years since the *Framework* was published. The accompanying text box describes key aspects of the proposed guidelines in more detail.

Following revision and publication of the final guidelines for ecological risk assessment, EPA plans to publish a series of shorter, more focused documents that address specific ecological risk assessment topics. As the Agency guidelines are finalized and supplemented by additional guidance, the framework and general approaches contained in them will be reviewed to see if additional guidance relevant to ecological risk assessment for air toxics might be provided.

### **PROPOSED GUIDELINES FOR ECOLOGICAL RISK ASSESSMENT**

The proposed guidelines – the first Agency-wide guidelines for ecological risk assessment – were developed to improve the quality of and consistency among EPA's ecological risk assessments (EPA 1996c). The proposed guidelines expand upon and replace the widely used EPA report, *Framework for Ecological Risk Assessment* (EPA 1992a). The proposed guidelines are intentionally broad in scope in order to cover the full range of ecological risk assessment problems and do not provide detailed guidance. The EPA intends to prepare more detailed guidance on more specific areas in the future. The content and focus of the proposed guidelines include the following.

- ▶ Risk assessors and risk managers at EPA are the primary audience for the document, although others outside the Agency (e.g., State agencies, and other interested parties) may find the proposed guidelines useful.
- ▶ Ecological risk assessment is defined as a process for organizing and analyzing data, information, assumptions, and uncertainties to evaluate the likelihood that one or more stressors are causing or will cause adverse ecological effects.
- ▶ Ecological risk assessments consist of three primary phases: problem formulation, analysis, and risk characterization.
  - Within the problem formulation phase, important areas include identifying goals and assessment endpoints; preparing a conceptual model; and developing an analysis plan.
  - The analysis phase involves evaluating exposure to stressors and the relationship between stressor levels and ecological effects.
  - In the risk characterization phase, key elements are estimating risk through integration of exposure and stressor-response profiles; describing risk by discussing lines of evidence and determining ecological adversity; and preparing a report of the risks.
- ▶ A major theme of the proposed guidelines is the interaction between risk assessors and risk managers at the beginning and end of the risk assessment process. Regarding problem formulation, the proposed guidelines emphasize the complementary roles of assessors and managers in determining the scope and boundaries of the assessment and selecting endpoints that will be the focus of the assessment. When the risk characterization is complete, the risk assessor must communicate the risks “in a manner that is clear, transparent, reasonable, and consistent” with Agency risk characterizations of similar scope. The guidelines specify that the risk assessor must discuss the results with the risk manager to facilitate the risk manager’s understanding of the major or potential risks and the risk assessment’s limitations. The interaction between risk assessors and risk managers is critical to ensure that the results of the assessment can be used to support a management decision. Ecological risk assessments may be conducted in sequential tiers that proceed from a simple, relatively inexpensive screening-level assessment based on conservative assumptions to more complex and costly assessments that require more refined analytical techniques and data. Higher tier risk assessments, although more costly, provide more ecologically realistic assessments and use less conservative assumptions.

### **2.3.5 State and Local Air Toxics Programs**

An additional component to risk assessment development has been the development of State and local air toxics programs and the interactions that EPA has had with these programs. Prior to passage of the 1990 CAA Amendments, the Federal air toxics program progressed slowly. In the absence of a strong Federal program, many State and some local agencies began to respond to the air toxics problem by developing their own programs. As a result, many States in the country currently have an air toxics control program in place addressing, at a minimum, new sources of toxic pollutants. Some have their own regulations that allow them to actively control air toxic emissions to a level protective of human health; others rely on comprehensive policies or authority provided to implement the Federal program. Some programs are risk-based, while others are technology-based (STAPPA/ALAPCO 1989).

The State and local programs have focused on three methods for addressing air toxic emissions: (1) ambient air levels; (2) control technology standards; and (3) risk assessment. Over

time, many have begun to use combination approaches, such as residual risk assessment which combines control technology and risk assessment. The main difference between the State/local residual risk assessment approach and the strategy set forth in sections 112(d) and 112(f) of the CAA is one of timing. While the CAA envisions control of HAPs from major sources as a two-step process (MACT followed by residual risk), with the two steps separated in time by as much as nine years, many State and local agencies generally consider, simultaneously, control technology and residual risk assessment. Both steps are generally completed within the context of a single permit application.

The State and local air toxics programs were invaluable prior to the CAA, and they remain invaluable. The EPA has drawn upon the expertise and experience of State and local agencies to assist in the development of the Federal risk program for HAPs. Over the years, more and more State and local air toxics programs have begun to use risk assessment, especially residual risk assessment. In a survey of State and local agencies, conducted in August of 1995, 60 percent of the respondents indicated that their air toxics program was risk-based, and 50 percent of those with residual risk programs addressing both new and existing sources.

Most State and local agencies that are currently using residual risk assessments plan to continue to use them for permitting purposes, so these may be available to EPA as residual risk assessments are prepared on a national basis. The EPA will identify the programs that are currently producing residual risk assessments, the situations in which they are produced, and the type of information contained in the permit applications or accompanying documents in order to add this information to the national residual risk assessment program.

The State and local programs have made progress in addressing the air toxics problem and protecting the health of their people and their environment. A successful residual risk program will be one which integrates the Federal program with the State and local programs and strengthens those existing programs. The Federal program will need to integrate these existing programs through the interactive sharing of expertise, data, analyses, and methodologies in order to ensure that human health and the environment are protected. Additionally, the State and local authorities may complement the Federal program by addressing local risk issues that may not be effectively addressed nationally.

### **3. Section 112 (f)(1)(A): Methods for Assessing Risks: EPA'S General Risk Assessment Approach for Air Toxics**

The information presented thus far provides the legislative and historical basis for the air toxics risk assessment process as it exists today. The EPA has refined the process over time using information from the reports discussed, information from and discussions with State, local, and regional air toxics risk assessors, and information and experience gathered from the practical application of risk assessments. This section describes the risk assessment process for air toxics that has developed at EPA. These risk assessment methods and policies will form the underlying basis for EPA's residual risk assessment strategy, which is described in Chapter 5. Section 3.1 addresses human health risk assessment methods, Section 3.2 addresses ecological risk assessment methods, Section 3.3 describes data needs for risk assessment, and Section 3.4 discusses assessment of mixtures.

#### **3.1 Human Health Risk Assessment of Air Toxics**

This section is organized according to the four basic risk assessment steps defined by NRC: hazard identification, dose-response assessment, exposure assessment, and risk characterization.

##### **3.1.1 Hazard Identification**

The first step in a risk assessment is to determine whether the pollutants of concern are causally linked to the health effects in question. This is the hazard identification. Factors such as the route of exposure, the type and quality of the effects, the biological plausibility of findings, the consistency of findings across studies, and the potential for bioaccumulation all contribute to the strength of the hazard identification statement. There are many sources of information that can be brought to bear in the hazard identification. The following text box summarizes the sources of information for hazard identification.

In performing the hazard identification and dose-response analysis, it is useful to divide the approaches into several categories of effects. The category(ies) of effects that are relevant to a particular chemical are determined as part of the hazard identification. The approaches for dose-response assessment and risk characterization differ for each category of effect. The distinct categories of health effects used or proposed for future use by EPA in air toxics risk assessment are:

- Non-cancer effects – chronic;
- Non-cancer effects – acute;
- Cancer, with linear extrapolation; and
- Cancer, with nonlinear extrapolation.

#### **SOURCES OF INFORMATION FOR HAZARD IDENTIFICATION**

**Epidemiologic data.** Studies of human populations exposed to HAPs in occupational settings or in the general environment can provide valuable information on the effects of HAPs. These studies have advantages over other sources of information in that they directly assess the effects of exposure to humans and, in the case of studies of the general population, address exposures that actually occur in the environment. In addition, recent work with biomarkers (chemicals in the body which allow for better quantification of exposure) promises to boost the utility of epidemiology in the future. Shortcomings include concerns about the relevance of high exposure levels often seen in occupational studies to environmental concentrations, concerns over the control of “confounding” variables (such as tobacco use) that may obscure true causal relationships (or imply false ones), difficulties in adequately characterizing exposure, and the difficulty most epidemiologic studies have in discerning subtle effects (see Section 4.2.1 for a more complete discussion of epidemiologic data in the context of section 112(f)).

**Animal toxicologic data.** High-quality studies of human populations exposed to HAPs are rare, due to both expense and the inherent limitations of epidemiology. As a result, EPA and others commonly rely on animal studies to infer potential risk to humans. Animal toxicologic data are typically much easier to obtain than good epidemiologic data, and effects can be explicitly linked with exposure to the HAP(s) being tested with little fear of confounding. However, issues of high-to-low-dose relevance are compounded by the need to extrapolate the effects seen in animals to those anticipated in humans. Although there have been considerable advances in understanding the relevance of specific results in animal studies to human biology, such extrapolations remain a considerable source of uncertainty. The EPA has operated under the conservative public health policy which assumes that adverse effects seen in animal studies indicate potential effects in humans.

**Short-term in vitro assays.** *In vitro* tests can be carried out quickly and at relatively low cost, and they can provide valuable information on specific aspects of a pollutant’s toxicity, such as a particular mechanism of mutagenicity that may be an initiating event for cancer. However, such tests typically provide only supporting information about a pollutant’s effects, as few tests have been developed that are specific to a particular effect or disease.

**Structure/activity relationships.** By comparing the molecular structure of a pollutant with that of others of known toxicity, toxic effects can sometimes be inferred, particularly if there is knowledge about the mechanism of action. This approach is often useful when examining the hazards associated with individual compounds within a class of related compounds (e.g., dioxins) or when identifying compounds for future study. Although structure/activity analyses are rarely a substitute for existing experimental or epidemiologic data, and represent a relatively uncertain basis for hazard identification, they are useful when experimental data are absent.

#### **Non-cancer Effects – Chronic and Acute**

In large part due to the wide variety of endpoints, hazard identification procedures are less formally set out for non-cancer effects than for the identification of carcinogens. The EPA has published guidelines for several specific types of non-cancer effects, including mutagenicity assessment (EPA 1986b) and developmental toxicity assessment (EPA 1991a), and also has published proposed guidelines for neurotoxicity assessment (EPA 1995b) and for reproductive toxicity assessment (EPA 1996d). Rather than specifying risk assessment methodology, the non-cancer guidelines tend to focus on the proper conduct of testing and the appropriate toxicological interpretation of results of the commonly performed assays. The guidance for hazard identification decisions is fairly general.

For assessment of chronic toxic effects other than cancer, EPA’s general approach to hazard identification is to review the health effects literature and characterize its strengths and

weaknesses, using primarily a narrative approach rather than a formal classification scheme. Available data on different endpoints are arrayed and discussed, describing the effects (and their attendant dose/exposure levels). While there may be no formal hierarchy, particular attention is given to effects that occur at relatively low doses or that may have particular relevance to human populations. The narrative description of the data base discusses factors such as the methodological strengths and weaknesses of individual studies (as well as the overall data base), the time period over which the studies were conducted (e.g., chronic vs. subchronic), routes of exposure, and possible biological mechanisms. In the course of this narrative, there is discussion of effects, which may range from severe frank effects that can cause incapacitation or death to subtle effects that may occur at the cellular level, but are early indicators of toxic effects. Not all effects observed in laboratory studies are subsequently judged to be adverse effects. The distinction between adverse and non-adverse effects is not always clear-cut, and considerable professional judgment is required in applying criteria to identify adverse effects. All of these observations are integrated into a presentation that gives a concise profile of the toxicological properties of the pollutant.

In addition to toxicity related to long-term exposures, many HAPs also cause toxic effects after short-term exposures lasting from minutes to several hours. Indeed, for some pollutants acute exposures are of greater concern than chronic exposures. While various EPA offices have addressed acute exposures across a variety of regulatory programs, Agency-wide guidance on how to assess toxic effects from short-term exposures had been lacking until recent publication of a draft acute methods document (EPA 1994c). This draft document, however, is still undergoing review, and therefore its use is limited. An interagency group including EPA currently is assessing hazard and developing quantitative values (referred to as acute exposure guidance levels, or AEGLs) for acute toxicity of specific chemicals, following guidance published by the NRC (NRC 1993b). See Section 3.3.1 for more discussion of various acute toxicity values.

### **Cancer**

The EPA's 1986 Guidelines for Carcinogen Risk Assessment (EPA 1986c) provide guidance on hazard identification for carcinogens. The approach recognizes three broad categories of data: (1) human data (primarily epidemiological); (2) results of long-term experimental animal bioassays; and (3) a variety of data on short-term tests for genotoxicity and other relevant properties, pharmacokinetic and metabolic studies, physio-chemical properties, and structure-activity relationships. In hazard identification of carcinogens under the 1986 guidelines, the human data, animal data, and "other" evidence are combined to characterize the weight of evidence regarding the agent's potential as a human carcinogen into one of several hierarchic categories. The text box below outlines these categories.

**WEIGHT-OF-EVIDENCE CATEGORIES IN EPA'S 1986 CANCER RISK GUIDELINES**

- ▶ **Group A Carcinogenic to Humans:** Applies when there are adequate human data to demonstrate the causal association of the agent with human cancer (typically epidemiologic data).
- ▶ **Group B Probably Carcinogenic to Humans:** Agents with sufficient evidence (i.e., indicative of a causal relationship) from animal bioassay data, but either limited (i.e., indicative of a possible causal relationship, but not exclusive of alternative explanations) human evidence (Group B1), or with little or no human data (Group B2).
- ▶ **Group C Possibly Carcinogenic to Humans:** Agents with limited animal evidence and little or no human data.
- ▶ **Group D Not Classifiable as to Human Carcinogenicity:** Agents without adequate data either to suggest or refute the suggestion of the human carcinogenicity.
- ▶ **Group E Evidence of Non-Carcinogenicity for Humans:** Agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies (EPA 1986c).

The EPA has proposed major revisions of the carcinogen hazard identification scheme. The proposed revision to the cancer risk assessment guidelines (EPA 1996b), which is expected to be finalized in 1998 and then used to guide the residual risk assessment, focuses on narrative statements describing the main lines of evidence and their interpretation, in place of the current pre-defined hierarchical categories with alphabetic designations. Rather than the three-step process used under the 1986 guidelines of separately evaluating human evidence, evaluating animal evidence, and combining these judgments into an overall weight of evidence (while considering the short-term test data), the proposed guidelines suggest a single comprehensive evaluation process. This process stresses the explicit consideration of coherence of the various data elements into one scientific interpretation that evaluates, to the extent possible, how well the commonality of mode of carcinogenic action between human beings and the various test systems has been established. Emphasis is also placed on defining the qualitative conditions under which carcinogenic hazards might be expected. If warranted, limitations to the finding of carcinogenic hazard can be drawn based on route of exposure, necessity of some other factors for which tumorigenesis is necessary, and doses below which elevation of cancer risk is not expected. Key differences between the 1996 proposed revised cancer guidelines and the original 1986 guidelines are highlighted in the accompanying text box.

### **3.1.2 Dose-response Assessment**

Dose-response assessment is the characterization of the relationship between the concentration, exposure, or dose of a pollutant and the resultant health or environmental effects. The nature of quantitative dose-response assessment varies among pollutants. Sufficient data often exist for criteria air pollutants, such as ozone or carbon monoxide, so that relatively complete dose-response relationships can be characterized. In such cases, there is no need for

**SUMMARY OF MAJOR DIFFERENCES BETWEEN EPA'S 1986 GUIDELINES (EPA 1986c) AND THE 1996 PROPOSED GUIDELINES FOR CARCINOGEN RISK ASSESSMENT (EPA 1996b)**

**1986 Guidelines**

**1996 Proposed Guidelines**

*Weighing Evidence of Hazard*

- |   |  |
|---|--|
| <ul style="list-style-type: none"><li>• Decisions are based almost exclusively on tumor findings in animals and/or humans.</li><li>• Human and animal evidence are evaluated separately and combined into the overall weight of evidence.</li></ul> | <ul style="list-style-type: none"><li>• Decisions take into account <u>all</u> available evidence (e.g., structure-activity relationships, mode of action).</li><li>• All data are evaluated in a single comprehensive evaluation process.</li></ul> |
|---|--|

*Classification Descriptors*

- |  |  |
|--|--|
| <ul style="list-style-type: none"><li>• Substance is assigned a weight of evidence classification (A through E) regarding its potential to cause cancer in humans.</li></ul> | <ul style="list-style-type: none"><li>• A narrative statement with descriptors (e.g., "known/likely" to be carcinogenic) is developed for a substance, and includes information on the lines of evidence, exposure pathways, conclusions, and limitations.</li></ul> |
|--|--|

*Dose-response Assessment*

- |  |   |
|--|---|
| <ul style="list-style-type: none"><li>• Default model used for linear dose-response relationships is the "linearized multistage" procedure.</li><li>• Dose-response evaluation is limited to carcinogenicity data.</li></ul> | <ul style="list-style-type: none"><li>• Biologically based dose-response models are used whenever data are sufficient. Recommended default approaches include the margin of exposure approach and linear extrapolation to zero dose, zero response.</li><li>• If appropriate, data on non-carcinogenic effects may be used to help characterize the carcinogenicity dose-response relationship.</li></ul> |
|--|---|

extrapolation to lower doses because adequate health effects data are available, often in humans, at environmental levels. Such is not the case for most air toxics. Typically, when adequate data exist, the most that can be identified in the dose-response assessment for hazardous air pollutants is a sub-threshold dose or exposure level that humans could experience daily for a lifetime without appreciable probability of ill effect. In general, the effects data base is reviewed to define a LOAEL or, preferably, a NOAEL from valid experimental or epidemiologic data, adjusted by inter-species scaling factors, if necessary. Uncertainty factors (UFs) that range individually from 3 to 10 are then applied to the NOAEL to account for such variables as inter-species susceptibility, sensitive human populations, and difference in exposure times. The result of this procedure, subject to peer review, is an RfD for oral (ingestion) exposure to an agent or an RfC for inhalation exposure. In addition to a numeric RfD or RfC, EPA also develops a degree of confidence statement (of either high, medium, or low). An alternative to the NOAEL procedure

is to identify specific features of the existing data such as an effective dose associated with a given level of response. Benchmark dose analysis, for example, may be used to estimate a dose associated with the lower bound on the 10 percent response to which UFs are then applied.

It should be noted that exposures above an RfD or RfC do not necessarily imply unacceptable risk or that adverse health effects are expected. The significance of such exceedances must be evaluated on a case-by-case basis, considering such factors as the confidence level of the assessment, the size of UFs used, the slope of the dose-response curve, the magnitude of the exceedance, and the number or types of people exposed at various levels above the RfD or RfC.

As noted earlier, most epidemiologic and toxicologic data on HAPs typically result from exposure levels that are high relative to environmental levels. When a quantitative dose-response relationship is desired for carcinogens, models are often employed to extrapolate from high to low doses. In the absence of valid data to the contrary, EPA has typically applied a linear non-threshold model to estimate cancer risks at low exposures. However, as stressed in the Proposed Guidelines for Carcinogen Risk Assessment (EPA 1996b), when there are adequate mechanistic data to suggest that other models would be more appropriate to estimate low-exposure risk, they may be used on a case-by-case basis. In the absence of such data, the assumption of response linearity is maintained although the modeling scheme has been simplified.

The “margin-of-exposure” (MOE) approach has been advocated recently as an alternative to the development of complete quantitative dose-response relationships (Proposed Guidelines for Carcinogen Risk Assessment, EPA 1996b; The Commission on Risk Assessment and Risk Management’s report, CRARM 1997b; see Section 2.3 for a more complete discussion). In this approach, the data are analyzed as with the benchmark dose in order to identify a specific feature such as an ED<sub>10</sub> (effective dose at the 10 percent response) or the lower bound on the ED<sub>10</sub>. This value is then compared directly with estimated exposures rather than having uncertainty factors applied.

In summary, dose-response assessment methods generally consist of two parts. First is the evaluation of data in the observable range, and second is the extrapolation from the observable range to low doses/risks. Recent terminology refers to the result of analysis in the observable range as the “point of departure,” from which extrapolation begins. The approaches used for evaluation in the observable range are similar for all three categories of effects (non-cancer, cancer linear, and cancer nonlinear), while the extrapolation methods for each differ considerably.

### **Non-cancer Effects    Chronic**

The inhalation RfC and oral RfD are the primary Agency consensus quantitative values for use in non-cancer risk assessment. The RfC/D is defined as an estimate, with uncertainty spanning perhaps an order of magnitude, of an inhalation exposure/oral dose to the human population (including sensitive subgroups) that is likely to be without appreciable risks of

deleterious effects during a lifetime. The RfC/D is derived after a thorough review of the health effects data base for an individual chemical and identification of the most sensitive and relevant endpoint and the principal study(ies) demonstrating that endpoint. As discussed under hazard identification in Section 3.1.1, not all effects that can be observed in studies are determined to be adverse effects; a non-adverse effect would not be selected as the critical effect on which to base an RfC/D. Inhalation RfCs are derived according to the Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA 1994b), which was developed by EPA. The RfC/D should represent a synthesis of the entire data array. The evaluation of and choice of data on which to base the RfC/D derivation are critical aspects of the assessment and require scientific judgment.

The derivation of the RfC/D begins with identification of adverse effects from available human or animal studies, followed by the identification of NOAELs and/or LOAELs. The LOAELs and NOAELs from animal studies are converted to Human Equivalent Concentrations (HEC) using dosimetric methods (described in EPA 1994b). The NOAEL[HEC] or LOAEL[HEC] from one or a few studies that is representative of the threshold region of observable effects is the key datum gleaned from evaluation of the dose-response data. The RfC/D is derived from the NOAEL[HEC] or LOAEL[HEC] by consistent application of UFs.

Recently, the benchmark dose (BMD)/benchmark concentration (BMC) approach has sometimes been used to effectively derive the LOAEL or NOAEL used as the basis for derivation of the RfC or RfD. The UFs are applied to account for recognized areas of uncertainty in the extrapolation from the experimental data and exposure conditions to the human lifetime exposure scenario. The standard UFs are applied as appropriate for the following extrapolations or areas of uncertainty:

- Laboratory animal data to humans;
- Average healthy humans to sensitive humans;
- Subchronic to chronic exposure duration;
- LOAEL to NOAEL; and
- Incomplete data base.

The UFs that are generally applied range from a factor of three to an order of magnitude. The composite UF will depend on the number of extrapolations required. RfCs have been derived using composite UFs that range from 10 to 3,000, with most RfCs using factors of 100 to 1,000. The UF for animal to human extrapolation in RfC development typically is less than an order of magnitude due to the dosimetric adjustments employed. It is also common that chemical-specific information is used to reduce the UF in other extrapolations. For example, the subchronic to chronic UF for acrylic acid was reduced because a comparison of two-week and 90-day studies showed minimal difference in the incidence or severity of effect, suggesting that there was little difference at various exposure durations. Likewise, the LOAEL to NOAEL extrapolation UF has been reduced for several RfC derivations because the effect at the LOAEL was very mild. In general, studies (e.g., Baird et al. 1996) have shown that the default UF of 10 may be

conservative in many cases, and the UF is therefore a key parameter for examination in uncertainty analyses. When reductions in the UF are used, a factor of three is used as a convention because it is a half-order of magnitude in log space (i.e.,  $10^{1/2}$ ) rounded to one significant figure. It is also common to reduce the composite UF when four areas of uncertainty are present, in recognition that it is unlikely that the different sources of uncertainty will all simultaneously be near maximum values.

The use of order of magnitude uncertainty factors, RfCs/Ds and UFs rounded to one significant figure, and the definition of the RfC/D as having “uncertainty, spanning perhaps an order of magnitude” are indications of the general lack of precision in the estimates. The uncertainty resulting from any single area of extrapolation is not well understood or precisely defined. Current efforts to develop more rigorous statistical descriptions of the uncertainty in extrapolating from, for example, animals to humans or subchronic to chronic exposures may lead to a probabilistic method for assigning UFs. The current state-of-the-art, however, relies on point estimates of uncertainty and therefore results in point estimates of the RfC/D. The individual UFs are generally considered to be somewhat conservative, if not reduced due to specific data. It follows that the greater the overall magnitude of the UF (i.e., the more individual UFs that were combined to get the total UF), the more conservatism is included. The precision of “an order of magnitude” should be considered to apply on the average. Less precision would be implied in the case of an RfC with a greater UF (e.g.,  $\geq 1,000$ ) and more precision would be suggested for RfCs with lower overall UFs (e.g.,  $\leq 100$ ). The relative precision and the magnitude of the composite UFs will be important considerations in decisions involving comparisons of hazard quotients for different chemicals and in assessing the hazard index (HI) for a mixture of chemicals.

Recently, the BMC/BMD approach has been used to supplement the approaches based on LOAELs and NOAELs. The BMD approach is an alternative to the NOAEL approach as a way to identify a dose without appreciable effect based on experimental data. The BMD approach fits a dose-response curve to the data in the observed experimental range. A lower bound on the dose causing some specified level of risk above background (e.g., 10 percent) is calculated, and this dose value is used as a point of departure for the application of UFs in place of the experimental NOAEL or LOAEL. That is, it is taken as a standardized measure of a dose level near that at which an experimental response would no longer be expected to be evident using standard study designs. The BMD considers the entire data set, accounts for the sample size, and does not depend on a data point as does the NOAEL. A primary problem with a NOAEL is the wide range of risk that may be present at the NOAEL depending on experimental design; the benchmark approach minimizes this problem. The BMD also can bring to bear information on the overall pattern of response, including the steepness of the dose-response relationship. The benchmark approach has been used by EPA in several recent RfC/D assessments.

#### **Non-cancer Effects    Acute**

EPA is currently developing a method for dose-response assessment of acute exposures that is substantially similar to the approach for chronic exposure. Similar approaches based on

applying uncertainty factors to acute toxicity data points (e.g., LOAEL,  $LEC_{10}$ , NOAEL) have been developed and used by various groups (see Section 3.3.1). In EPA's new method, however, in addition to the use of either a LOAEL or NOAEL, or a BMD, a third approach to dose-response analysis is used that is particularly amenable to the available data for acute exposure for many chemicals. The third approach is referred to as categorical regression and allows the combination of data from different studies in order to evaluate the role of both exposure concentration and duration in producing the effect (EPA 1994c). Data are combined by expressing various effects on a common scale of severity and performing a regression analysis of severity vs. concentration and duration. The results of a categorical regression analysis are used in the same way as a BMC/D or a NOAEL, as the departure point for extrapolation to the human exposure of interest. In the case of the NOAEL or the BMC, the departure point is a point estimate. In categorical regression, the departure point can be a line on a concentration vs. time plot, with the result that any duration of acute exposure can be interpolated along that line. The line is actually a composite of likelihood estimates calculated from the regression results. For example, a concentration-time line indicating the 10 percent likelihood of observing a specific category of effect, termed an  $ECT_{10}$  line, could be generated that is analogous to a  $BMD/C_{10}$ . The appropriate approach for dose-response analysis will depend on the amount and quality of the available data. In general, the NOAEL, BMC, and categorical regression techniques have increasing data requirements, so the most appropriate approach will be dictated by the available data. Once the best estimate of a point of departure is determined, the derivation of the acute reference exposure (ARE) proceeds with the consistent application of UFs. In general, the point of departure from the BMC approach and categorical regression is considered to represent an estimate of a NOAEL or LOAEL, depending on the level of risk associated with the BMC. The appropriate UFs will then include factors for extrapolation from animals to humans and from average to sensitive humans. An additional factor may be applied for incomplete database concerns.

## **Cancer**

The EPA's cancer risk assessment guidelines of 1986 adopted a default assumption that chemical carcinogens would exhibit risks at low doses. Extrapolation of cancer risk using the linearized multistage model, which results in a linear extrapolation of risk in the low dose region, was proposed as a reasonable upper bound on risk, and this approach has been used for most chemicals with adequate data since then. If animal data are used in the dose-response assessment, a scaling factor based on the surface area of the test animals relative to humans is used to calculate a human equivalent dose. Surface area is used for this scaling because it is a good indicator of relative metabolic rate.<sup>2</sup> Dose-response models such as the multistage model have

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<sup>2</sup> As specified in *Federal Register* 57(109):24152-24173 (June 5, 1992), "in the absence of adequate information on pharmacokinetic and sensitivity differences among species, doses of carcinogens should be expressed in terms of daily amount administered per unit of body mass raised to the 3/4 power. Equal doses in these units (i.e., in  $mg/kg^{3/4}/day$ ), when experienced daily for a full lifetime, are presumed to produce equal lifetime cancer risks across mammalian species." This scaling method is assumed to be intermediate between scaling by body mass and scaling by body surface area.

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historically been used to calculate upper-bound unit risk estimates (URE). Typically, EPA has relied on the URE as a quantitative measure of potential cancer hazard. A URE represents an estimate of the increased cancer risk from a lifetime (70-year) exposure to a concentration of one unit of exposure. The URE for inhalation exposures is typically expressed as risk per  $\mu\text{g}/\text{m}^3$  for air contaminants. The URE is a plausible upper-bound estimate of the risk (i.e., the risk is not likely to be higher but may be lower and may be zero).

Since the publication of the original guidelines, however, considerable new knowledge has been developed regarding the processes of chemical carcinogenesis and the evaluation of human cancer risk. Currently, a revision of the cancer guidelines is in preparation which represents a considerable departure from the original guidelines. A fundamental and important advance in the proposed revision to EPA's cancer guidelines is the distinction between linear and nonlinear modes of action. The cancer data in the observable range are analyzed using a dose-response model similar to the models used in the BMC approach for non-cancer effects. The  $\text{LED}_{10}$  (the 95 percent lower confidence limit on dose associated with the estimated 10 percent increase in tumor or tumor-related response) is proposed as a possible point of departure for extrapolation although other options are being considered. The method of extrapolation from the point of departure differs depending on whether the assessment of the available data on the mode of action of the chemical indicates a linear or nonlinear mode of action. A linear extrapolation is generally appropriate when the evidence supports a mode of action of gene mutation due to direct DNA reactivity or another mode of action that is thought to be linear in the low-dose region. For linear extrapolation, a straight line is drawn from the point of departure to the origin, and the risk at any concentration is determined by interpolation along that line. A linear mode of action also will serve as a default when available evidence is not sufficient to support a nonlinear procedure, even if there is no evidence for DNA reactivity.

An assumption of nonlinearity is used when there is sufficient evidence to support a nonlinear mode of action. A nonlinear mode of action could involve a dose-response pattern in which the response falls much more quickly than linearly with dose, but still indicating risk at low doses. Alternatively the mode of action may theoretically have a threshold if, for example, the cancer response is a secondary effect of toxicity or an induced physiological change which is a threshold phenomenon. In most cases, EPA will not try to distinguish between modes of action with a "true threshold" and those that are nonlinear through the origin, because data are rarely sufficient to make this determination. Also, as a default science policy, nonlinear extrapolation to low doses will not be performed because there is no current basis to choose a model or determine the shape of the dose-response function. However, as more specific information on a HAP's mechanism of action becomes available and the data are sufficient to support the use of alternative models, EPA will use them.

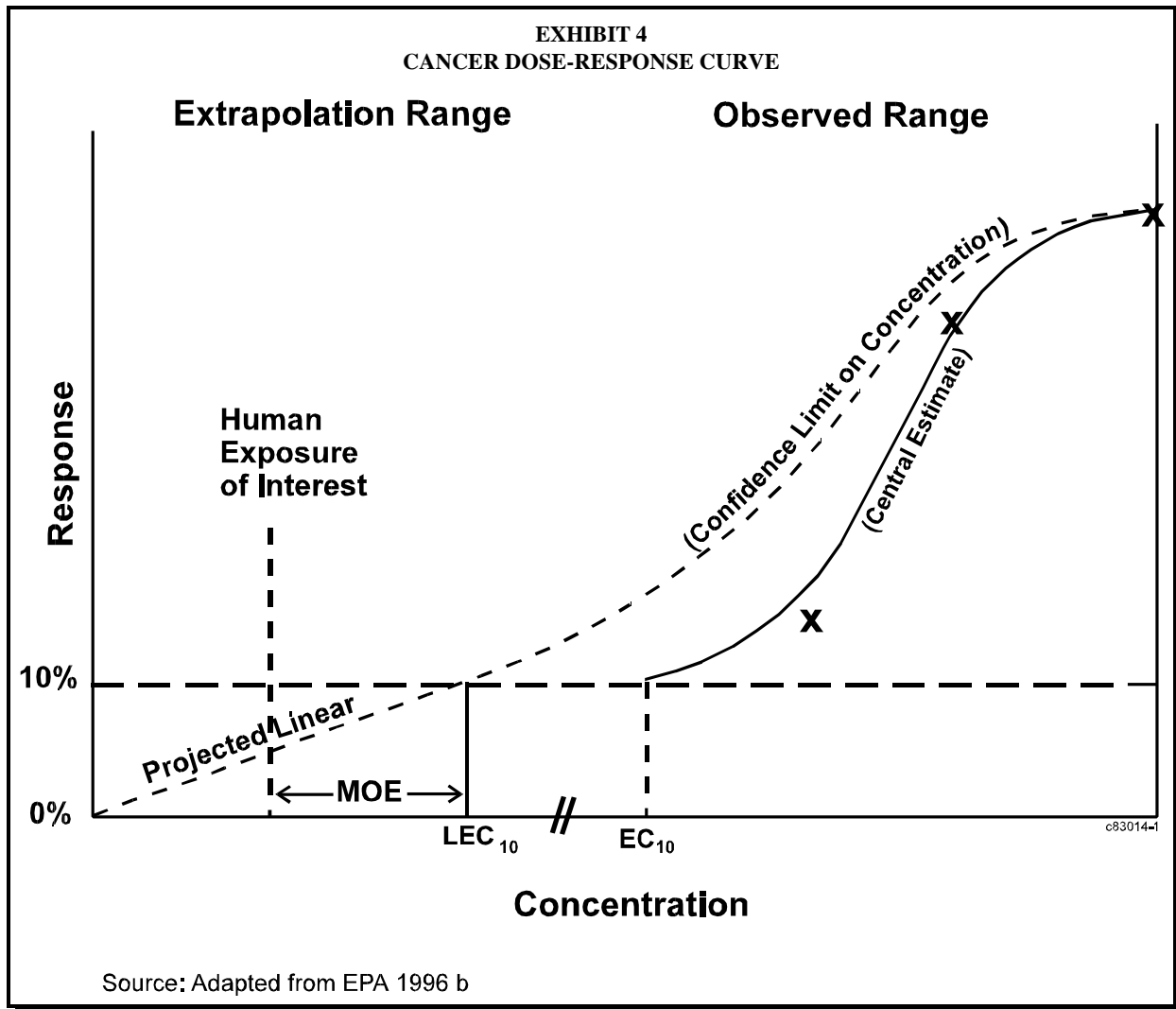
For carcinogens with nonlinear modes of action, an MOE approach has been proposed to evaluate concern for actual or projected exposure levels. In the proposed MOE approach, the point of departure from the available toxicity data is compared with the exposure level, and information is provided about the current understanding of the phenomena that may be occurring

as exposure decreases below the observed data. It is possible that the point of departure will be based on effects other than tumor data if, for example, the cancer response is determined to be secondary to a non-cancer effect.

Additional dose-response information will also be supplied to the risk manager. The information should include points such as the slope of the dose response curve, the nature of the response, the human variability in sensitivity, persistence of the agent in the body, and relative sensitivity of humans and animals. The point of providing related information is to allow the risk manager to consider all aspects of the data to inform the decision about the appropriate margin of exposure and the amount of reduction in risk associated with reduction in exposure below the point of departure. The endpoints relevant to the cancer assessment are determined based on a review of all relevant data.

As a default assumption, factors of 10-fold for human variability and species differences are proposed in the new cancer guidelines, if specific data do not indicate otherwise. These factors are not intended to be used as a default for an acceptable MOE, since each case should be considered individually and all hazard and dose-response factors should be considered together.

**Linear Extrapolation.** The proposed dose-response approach for cancer-causing agents for which there is evidence of direct-acting genotoxicity is to model the data in the observable range to determine the  $LEC_{10}$ . The only difference between this approach and the BMC approach for non-cancer effects is that the cancer modeling may be done using a single default approach, rather than the evaluation of several models and statistical comparisons to determine the best-fitting model as currently proposed for non-cancer endpoints. Using the  $LEC_{10}$  as the point of departure, the low-concentration extrapolation is done by extending a straight line from the  $LEC_{10}$  to zero dose and zero risk (the origin). The risk at any exposure concentration is then determined using that line. **Exhibit 4** depicts the linear cancer dose-response curve being discussed. The linearity assumption implies, among other things, that some risk exists at low doses and that risks from multiple chemicals are additive.



**Nonlinear Extrapolation.** The proposed approach is to model the data in the observable range in the same way as for linear carcinogens. Extrapolation from the  $LEC_{10}$  would involve an MOE analysis in which various other types of data would be considered to determine whether there is an adequate margin between the estimated exposures and the  $LEC_{10}$ . This approach is qualitatively different than the linear extrapolation described above because the explicit consideration of exposure estimates moves it into the realm of risk characterization. Exhibit 4 also depicts the MOE approach being discussed.

### 3.1.3 Exposure Assessment

The nature and complexity of the exposure assessment is often a function of the particular risk management question (or other purpose) to be addressed. Simple screening analyses, using conservative default assumptions, may be appropriate to rule out the need for further analyses or

action. On the other hand, a detailed exposure analysis may be necessary to determine the necessity or type of emission controls, particularly when those controls are associated with large economic consequences. In some cases, the critical policy question may be to estimate the risks to a small subset of the population at high exposure levels, whereas in another, the overall risks across the entire nation may be the driving policy question. Thus, there is no single "right" way to conduct an exposure assessment.

### **Agency Guidelines**

The EPA issued the *Guidelines for Exposure Assessment* on September 24, 1986 (EPA 1986a) and the *Proposed Guidelines for Exposure-related Measurements* on December 2, 1988 (EPA 1988b). In response to recommendations from the EPA Science Advisory Board and the public, the 1986 Guidelines were updated and combined with the 1988 Proposed Guidelines and reissued as the *Guidelines for Exposure Assessment*, which were published in final form on May 29, 1992 (EPA 1992c).

The *Guidelines for Exposure Assessment* were designed to aid risk assessors at EPA, and those consultants, contractors, or other persons who perform exposure and risk assessments under Agency contract or sponsorship. Publication of the Guidelines made information on the principles, concepts, and methods used by the Agency available to all interested members of the public. The Guidelines established a broad framework for Agency exposure assessments by describing the general concepts of exposure assessment, including definitions and associated measurement units, and by providing broad guidance on the planning and conducting of an exposure assessment. The Guidelines also provided information on presenting the results of the exposure assessment and characterizing uncertainty. Although the Guidelines focus on exposure of humans to chemical substances, much of the guidance also pertains to assessing wildlife exposure to chemicals, or to human exposures to biological, noise, or radiological agents.

In the Guidelines, EPA established a specific definition of exposure to minimize ambiguity in the use of terms and units for quantifying exposure. Human exposure is defined in the Guidelines as contact with a chemical or agent at the visible external boundary of a person, including skin and openings into the body such as mouth and nostrils (but not necessarily contact with exchange boundaries where absorption may take place, such as skin, lung, and gastrointestinal tract). Therefore, an exposure assessment is the quantitative or qualitative evaluation of contact, and includes such characteristics as intensity, frequency, and duration of contact. Often, an assessment also will evaluate the rate and route at which a chemical crosses the external boundary (dose) and the amount absorbed (internal dose). The numerical output of an exposure assessment may be either exposure or dose, depending on the purpose of the evaluation.

## **Components of Exposure Assessment**

An exposure assessment has four major components: (1) emissions characterization; (2) environmental fate and transport; (3) characterization of the study population; and (4) exposure calculation (EPA 1993a). In the emissions characterization component of exposure assessment, EPA collects data on emission rates of the pollutants and defines the parameters of the source. For point sources of air toxics, source parameters can include the flow rate of the stack gas volume, the stack gas exit temperature, and the stack height, among others. Source parameters define how the pollutant is released to the environment, and they affect the initial dispersion of the pollutant in the atmosphere. The fate and transport component, using the emission rate as a starting point, describes how the pollutant is ultimately transformed and dispersed over the area of interest. Transport and possible transformation of an airborne pollutant are influenced by the pollutant's physical and chemical properties, and by meteorological and environmental conditions. The population characterization component defines the study population in terms of geographic distribution and other characteristics of interest. Factors such as age, sex, and activity level affect the amount of pollutant actually inhaled by an individual, while mobility affects the concentration levels to which an individual is exposed over time. In the exposure calculation component, the pollutant concentration and study population are spatially integrated to estimate exposure (EPA 1993a).

The first step in an exposure assessment for air toxics is to determine the specific HAPs emitted and their sources of emission into the air. Depending on the analysis, these data can be derived from broad-scale emission inventories, specific data collection efforts with particular industries, or information from State or local air toxics agencies. Other information, such as the geographic location of release points, the temporal pattern of emissions (e.g., periodic "puffs" vs. constant emission rates), and the release height may be necessary depending on the level of detail needed in the assessment.

After the sources of HAPs have been identified, air dispersion models are often used to estimate air pollutant concentrations in the ambient air. The model chosen must be appropriate for the intended job, which may vary between estimates of short-term peak concentrations immediately adjacent to a facility, long-term concentrations over a city-wide area, or deposition over hundreds of miles. The HAP reactivity and persistence will influence dispersion as well and can be important factors in estimating exposure for certain pollutants. High-quality meteorological information is often crucial to a valid exposure assessment for air toxics, as well as information on local topography. The HAP monitoring data can be used either to check the validity of modeled concentration estimates or as the primary source of information for the exposure assessment itself.

The EPA currently uses the Human Exposure Model (HEM) (EPA 1986e) to estimate inhalation exposure from stationary sources of hazardous air pollutants. For more refined assessments, more sophisticated techniques using detailed, site-specific information can be used. Some of these techniques are being developed for residual risk assessment by EPA. In the

interim, the HEM contains meteorological data, census data, an EPA air dispersion model, and to address population activities and the variability associated with exposure assessment, an add-on Monte Carlo simulation routine. Industrial Source Complex Short-term 3, or ISCST3, a Gaussian plume model that can be used to estimate both short-term peak and long-term average air concentrations and deposition rates, also can be used in conjunction with HEM. Simpler EPA models, such as SCREEN3, may also be appropriate for screening-level air dispersion modeling. Various approaches to atmospheric fate and transport and exposure modeling are presented in *A Tiered Modeling Approach for Assessing the Risks Due to Sources of Hazardous Air Pollutants* (EPA1992b). The Assessment System for Population Exposure Nationwide (ASPEN), a model developed for EPA's cumulative exposure project, may also be able to be adapted and used for certain screening-level exposure assessments in residual risk analyses.

Predictions of ambient concentrations and atmospheric deposition derived from the HEM have not been validated. Model validation is a difficult, resource intensive process that relies heavily on monitoring data, and often the HEM predicts concentrations that are below the levels that can be detected using current analytic methods. Nevertheless, EPA continues to seek to improve the HEM by enhancing its capacity to incorporate exposure assessment tools and exposure data bases. For example, over the past decade the Agency has developed more sophisticated dispersion and exposure models and has significantly expanded available data bases on human activity patterns, breathing rates, residential occupancy periods, and microenvironmental exposures. The outputs of these improved dispersion models can be used as inputs to the HEM, along with more detailed and realistic exposure profiles, to generate better estimates of individual and population risk.

Likewise, ASPEN has not been validated. Unlike the HEM, however, ASPEN should be viewed as a potential modeling approach still under development. For many HAPs of interest, ASPEN's predicted concentrations span orders of magnitude. Comparisons between observed and predicted concentrations for carbon monoxide and benzene indicate that ASPEN currently has limited predictive capability. [See, for example, the presentation on ASPEN made to the Advisory Council on Clean Air Act Compliance Analysis on February 5, 1998.] EPA is making a number of modifications in the model intended to overcome these limitations, including updated estimates of emission inventories and efforts to correlate modeled source categories with MACT source categories. Finally, EPA is adding an exposure model that will account for a variety of important sources of risk variability, such as human activity patterns, commuting, and microenvironmental conditions. Whether ASPEN will ultimately prove useful as a residual risk screening tool will depend on the extent to which these modifications prove successful.

When ambient concentrations have been derived, the next concern is how to relate these to exposures. The locations of resources, homes, workplaces, schools, and other receptor points will partially determine the extent of actual exposure. For screening studies, an estimate of the maximum offsite concentration could be used to estimate potential exposure, while a refined assessment may require information about actual receptor points and the population's movement throughout the area, the amount of time spent in specific microenvironments (e.g., indoors at

home, outdoors, in motor vehicles), and building ventilation rates. For some studies, the most highly exposed 5 to 10 percent of the population may need to be well-characterized, while for others, the distribution of exposures across a wider area is desirable. Information on specific sensitive populations, such as children or the elderly, may also be desirable.

In recent years, there has been increasing interest in explicitly characterizing the extent of uncertainty and variability in risk assessment, and especially in the exposure assessment component. To do this, many assessors have turned to a technique known as Monte Carlo simulation analysis. Using this technique, important variables in the exposure assessment (as well as in the other parts of the risk assessment) are specified as distributions according to what can be expressed about their underlying variability and/or uncertainty. Variables are sampled repeatedly from these distributions and combined in the analysis to provide a range of outcomes. While this technique can offer a useful summary of complex information, it must be noted that the analysis is only as good as the underlying data. Assessors must take care that the individual modeled variables are expressed in a way consistent with the best information available, or the results of the Monte Carlo analysis will do more to confuse than enlighten.

The EPA has begun to develop the Total Risk Integration Methodology (TRIM), which is a multimedia, multipathway computer modeling system being designed to address all dimensions of a complete residual risk evaluation, including the exposure assessment. The TRIM will provide a framework for assessing human health and ecological risks from exposure to hazardous and criteria air pollutants. It will allow for the evaluation of multipathway exposure to air pollutants, using a dynamic mass-balance approach to estimate the exposure and dose profiles received by selected receptors. Both uncertainty and variability will be explicitly treated within the model framework. The TRIM should be available for EPA use by the year 2000.

### **Non-inhalation Exposures**

Many studies indicate that pollutants emitted into the atmosphere are passed to humans through non-inhalation pathways (EPA 1990a). An example would be a HAP depositing from the air onto the soil, followed by ingestion of the soil by a child. In actuality, for some HAPs, greater exposures to the HAP occur through non-inhalation exposures than through inhalation exposures. Much of the focus by EPA in conducting non-inhalation exposure assessments has centered around which pathways to address and which pollutants are the most likely to result in significant exposure.

Certain HAPs pose a particular concern for non-inhalation exposure, and these substances generally have common characteristics. They typically are persistent in the environment, have a strong tendency to bioaccumulate, and exhibit moderate to high toxicity.

The EPA's initial detailed guidance on multipathway exposure assessment methods was issued by ORD in 1990 (EPA 1990a), updated a few years later (EPA 1993b), and recently consolidated and updated again (EPA 1997i). These documents present the Indirect Exposure

Model (IEM), which consists of equations and default input values to be used in calculating exposure levels for a set of multipathway exposures. A critical input to these calculations is the HAP deposition rate (i.e., amount per unit time being deposited from the air to land and/or surface water) for the location(s) being assessed, which can be estimated using air models such as EPA's Industrial Source Complex, Short-Term (ISCST3).

In its initial guidance on hazardous waste combustion risk assessment (EPA 1994a), which built on the prior ORD guidance, EPA's Office of Solid Waste and Emergency Response identified the six pathways listed in **Exhibit 5** as potentially significant routes of non-inhalation exposure. Some pathways are only applicable for specific receptors (e.g., subsistence fisher), but other pathways are applicable for all four human receptors included in the guidance (adult, child, subsistence farmer, subsistence fisher).

**EXHIBIT 5**  
**NON-INHALATION EXPOSURE PATHWAYS INCLUDED IN**  
**OSWER'S 1994 COMBUSTION GUIDANCE**

Air  $\xrightarrow{\text{deposition}}$  Soil  $\xrightarrow{\text{ingestion}}$  Human

Air  $\xrightarrow{\text{deposition} + \text{uptake of vapor phase}}$  Above-ground Vegetable  $\xrightarrow{\text{ingestion}}$  Human

Air  $\xrightarrow{\text{deposition}}$  Soil  $\xrightarrow{\text{uptake of pore water}}$  Root Vegetable  $\xrightarrow{\text{ingestion}}$  Human

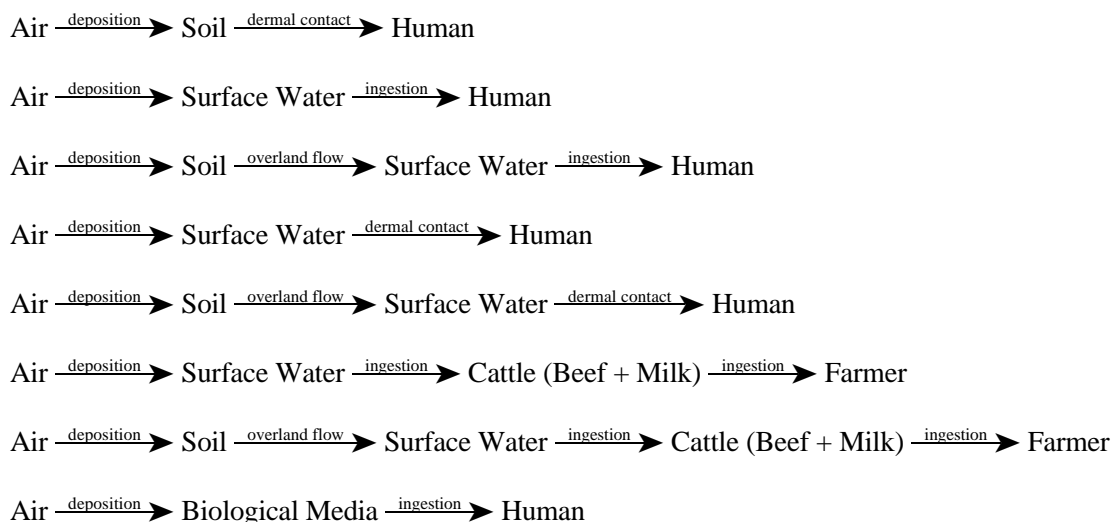
Air  $\xrightarrow{(\text{see above})}$  Soil + Above-ground Vegetable  $\xrightarrow{\text{ingestion}}$  Beef  $\xrightarrow{\text{ingestion}}$  Subsistence Farmer

Air  $\xrightarrow{(\text{see above})}$  Soil + Above-ground Vegetable  $\xrightarrow{\text{ingestion}}$  Milk  $\xrightarrow{\text{ingestion}}$  Subsistence Farmer

Air  $\xrightarrow{\text{deposition} + \text{runoff} + \text{erosion}}$  Waterbody  $\xrightarrow{\text{bioaccumulation}}$  Fish  $\xrightarrow{\text{ingestion}}$  Subsistence Fisher

As part of the proposed Hazardous Waste Identification Rule (HWIR), currently being developed by the Office of Solid Waste to identify levels of constituents in wastes that pose a low enough risk that Federal regulation as hazardous wastes is unnecessary, EPA designed a set of exposure pathways that was used in calculating low-risk concentrations for constituents in waste (EPA 1995c). Pathway design was built upon previous efforts by the Agency, and resulted in 30 human exposure pathways and 22 ecological exposure pathways. Fourteen pathways involve release to the air from the waste management unit and subsequent non-inhalation exposure to a receptor. Several of these 14 pathways are different from those listed in Exhibit 5, and they are presented in **Exhibit 6**.

**EXHIBIT 6**  
**ADDITIONAL NON-INHALATION EXPOSURE PATHWAYS INCLUDED IN HWIR**



EPA's Region 6 is currently refining the existing EPA guidance on hazardous waste combustion risk assessment (EPA 1997c). This draft guidance recommends consideration of the pathways listed in Exhibits 5 and 6, with the following changes.

- The draft guidance document states that historically the following pathways have been shown to be insignificant in the overall risk from combustor emissions and are therefore not recommended: groundwater ingestion, inhalation of resuspended particulate matter, and dermal exposure to surface water, soil, and air. The document does, however, recognize that in certain site-specific instances, it may be appropriate to include some of these pathways in the final risk assessment. In addition, groundwater uptake into food crops and livestock is considered minimal because of the hydrophobic nature of most bioaccumulative compounds.
- The updated guidance adds two new human receptors to the list: subsistence farmer child and subsistence fisher child. These two additional receptors receive exposure through the same pathways as their adult counterparts.
- If any of the following types of farms are found within the area of impact (or are expected to be located there in the future), then ingestion of the respective product should be added as a route of exposure: homegrown poultry, eggs from homegrown poultry, homegrown pork, and/or fish (from fish farming).

- The draft guidance recommends including infant exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalent (TEQ) through breast milk. This receptor and pathway are as follows:

Air  $\xrightarrow{\text{all inhalation and non-inhalation exposures}}$  Mother's Breast Milk  $\xrightarrow{\text{ingestion}}$  Infant

Exposures to other lipophilic compounds are also being discussed and may be addressed in future EPA guidance.

- Other sensitive subpopulations, if found in the area of impact, are to be modeled only for the direct inhalation pathway. These receptors may include pregnant women, the elderly, the infirm, and children in institutions (schools).

These draft recommendations, along with the entire updated hazardous waste combustion guidance, are currently under EPA review; an external draft should be available in the summer of 1998.

### **3.1.4 Risk Characterization**

The final step in the risk assessment process is the risk characterization, in which all the information from the previous steps is integrated to describe the outcome of the analysis. EPA's 1995 *Guidance for Risk Characterization* (EPA 1995a) lists two sets of guiding principles for defining risk characterization in the context of risk assessment. The following text box on risk characterization principles presents the three principles with respect to the information content and uncertainty aspects of risk characterization.

#### **RISK CHARACTERIZATION PRINCIPLES WITH RESPECT TO INFORMATION CONTENT AND UNCERTAINTY**

1. **The risk characterization integrates the information from the hazard identification, dose-response, and exposure assessments, using a combination of qualitative information, quantitative information, and information regarding uncertainties.** A good characterization should include different kinds of information from all portions of the foregoing assessment, carefully selected for reliability and relevance.
2. **The risk characterization includes a discussion of uncertainty and variability.** The risk assessor must distinguish between variability (arising from true heterogeneity) and uncertainty (resulting from a lack of knowledge).
3. **Well-balanced risk characterizations present risk conclusions and information regarding the strengths and limitations of the assessment for other risk assessors, EPA decision-makers, and the public.** "Truth in advertising" is an integral part of the characterization, discussing all noteworthy limitations while taking care not to become mired in analyzing factors that are not significant.

Similarly, several guiding principles are expressed in the guidelines with respect to various risk descriptors. In many ways, these descriptors tie into the way in which the exposure

assessment was conducted. The accompanying text box presents the five guiding principles with respect to various risk descriptors. Attention to these two sets of risk characterization principles will help ensure that the overall risk assessment is clear, honest, and does not overstep the bounds into risk management.

On May 15, 1997, EPA issued a document entitled *Policy for Use of Probabilistic Analysis in Risk Assessment* (EPA 1997b). It also issued an accompanying document entitled *Guiding Principles for Monte Carlo Analysis* (EPA 1997a). The policy and guiding principles are designed to support the use of various techniques for characterizing variability and uncertainty, a critical part of a complete risk characterization. The policy establishes conditions that are to be satisfied by risk assessments that use probabilistic techniques. These conditions relate to the good scientific practices of clarity, consistency, transparency, reproducibility, and the use of sound methods. The accompanying text box provides the conditions for an acceptable risk assessment that uses probabilistic analyses techniques. EPA's position, as stated in these documents, is "that such probabilistic analysis techniques as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessments."

**GUIDING PRINCIPLES WITH RESPECT TO RISK DESCRIPTORS**

1. **Information about the distribution of individual exposures is important to communicating the results of a risk assessment.** Both high-end and central tendency descriptors are used to convey the variability in risk levels experienced throughout the population.
2. **Information about population exposure leads to another important way to describe risk.** Both a probabilistic number of cases (or environmental impacts) and an expected percentage of the exposed population (or ecological resource) with risk greater than a certain level are valuable ways to present information.
3. **Information about the distribution of exposure and risk for different subgroups of the population are important components of a risk assessment.** Highly susceptible individuals or areas should be identified as well as those highly exposed, when possible.
4. **Situation-specific information adds perspective on possible future events or regulatory options.** Consideration of alternative scenarios when conducting risk assessment can aid in risk management decisions.
5. **An evaluation of the uncertainty in the risk descriptors is an important component of the uncertainty discussion in the assessment.** Both quantitative and qualitative evaluations of uncertainty can be useful to users of the assessment and should be presented separate from variability.

**CONDITIONS FOR AN ACCEPTABLE RISK ASSESSMENT  
THAT USES PROBABILISTIC ANALYSIS TECHNIQUES**

- (1) The purpose and scope of the assessment should be clearly articulated in a “problem formulation” section that includes a full discussion of any highly exposed or highly susceptible subpopulations evaluated (e.g., children, the elderly, etc.). The questions the assessment attempts to answer are to be discussed and the assessment endpoints are to be well defined.
- (2) The methods used for the analysis (including all models used, all data upon which the assessment is based, and all assumptions that have a significant impact upon the results) are to be documented and easily located in the report. This documentation is to include a discussion of the degree to which the data used are representative of the population under study. Also, this documentation is to include the names of the models and software used to generate the analysis. Sufficient information is to be provided to allow the results of the analysis to be independently reproduced.
- (3) The results of sensitivity analyses are to be presented and discussed in the report. Probabilistic techniques should be applied to the compounds, pathways, and factors of importance to the assessment, as determined by sensitivity analyses or other basic requirements of the assessment.
- (4) The presence or absence of moderate to strong correlations or dependencies between the input variables is to be discussed and accounted for in the analysis, along with the effects these have on the output distribution.
- (5) Information for each input and output distribution is to be provided in the report. This includes tabular and graphical representations of the distributions (e.g., probability density function and cumulative distribution function plots) that indicate the location of any point estimates of interest (e.g., mean, median, 95th percentile). The selection of distributions is to be explained and justified. For both the input and output distributions, variability and uncertainty are to be differentiated where possible.
- (6) The numerical stability of the central tendency and the higher end (i.e., tail) of the output distributions are to be presented and discussed.
- (7) Calculations of exposures and risks using deterministic (e.g., point estimate) methods are to be reported if possible. Providing these values will allow comparisons between the probabilistic analysis and past or screening-level risk assessments. Further, deterministic estimates may be used to answer scenario-specific questions and to facilitate risk communication. When comparisons are made, it is important to explain the similarities and differences in the underlying data, assumptions, and models.
- (8) Because fixed exposure assumptions (e.g., exposure duration, body weight) are sometimes embedded in the toxicity metrics (e.g., reference doses, reference concentrations, unit cancer risk factors), the exposure estimates from the probabilistic output distribution are to be aligned with the toxicity metric.

Source: EPA 1997a

The NRC, in its recent review of EPA's risk assessment methodology for HAPs (NRC 1994), recommended that uncertainty and variability should be quantified and the distinction between uncertainty and variability maintained throughout the assessment. A model under development by EPA, TRIM, will do this explicitly. In the interim, a Monte Carlo assessment is sometimes conducted on the risk estimates produced by HEM. At present, such assessments primarily address variability, while uncertainty is largely described qualitatively. The variability assessment considers variation in such factors as the number of years residents occupy their primary residences, number of hours per day people are at home, breathing rates across the exposed population, the amount of ambient pollution that infiltrates to the indoor microenvironment, and certain meteorological variables. Thus, the results of the assessment may

be expressed in probabilistic terms, potentially providing the risk manager and the affected public with more information than was previously provided. However, care must be taken in the interpretation of such analyses, as they are only as reliable as the underlying data and assumptions. Uncertainty in risk assessment is discussed further in Section 4.2.3.

### **3.2 Ecological Risk Assessment of Air Toxics**

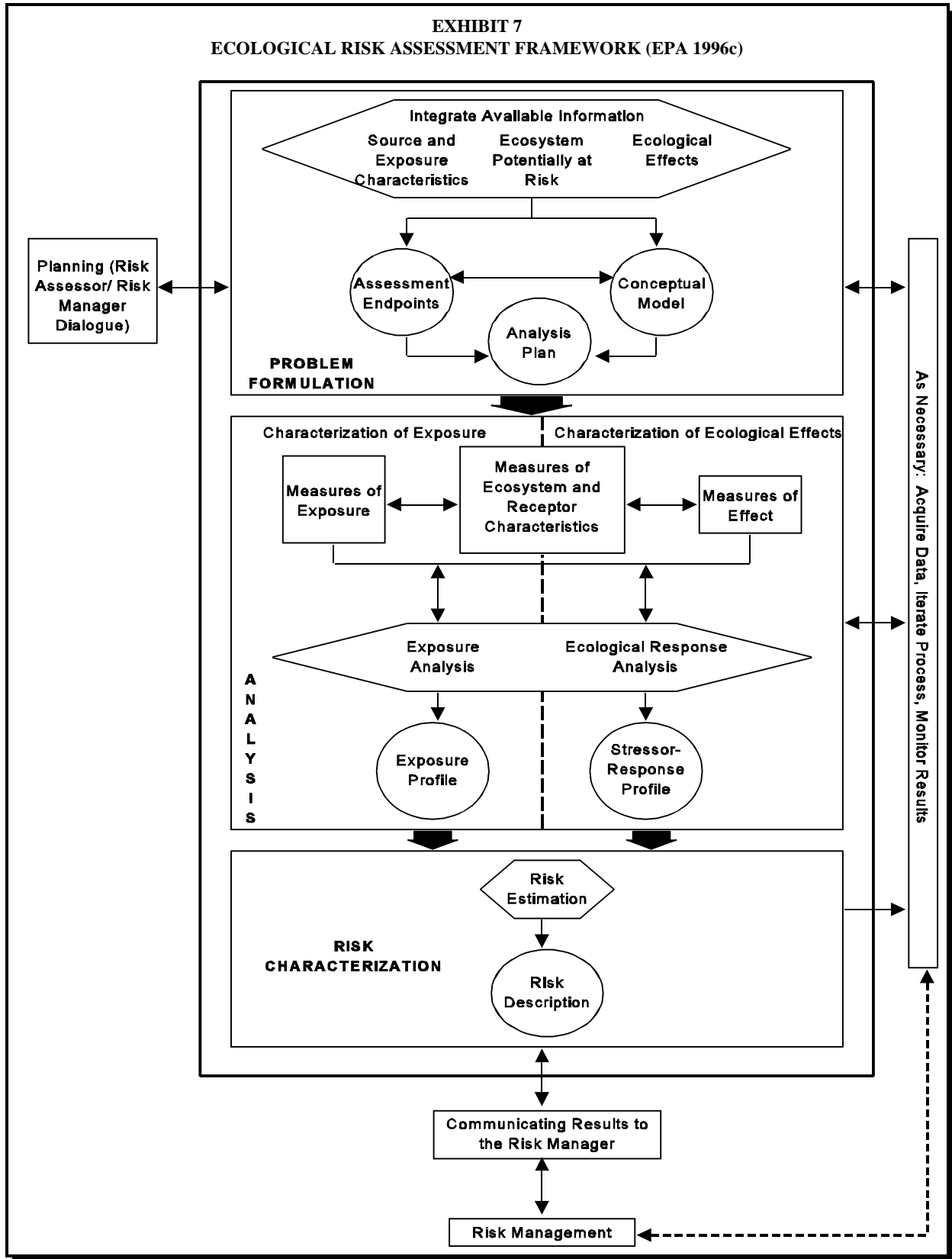
As discussed in Section 2.2 of this report, section 112(f)(2) of the CAA authorizes EPA to consider adverse environmental effects in developing residual risk standards. The statute directs EPA to promulgate standards that provide an ample margin of safety to protect public health, unless “a more stringent standard is necessary to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect.” Thus, assessing the ecological risks of HAPs will be a critical part of the residual risk program. This section describes EPA’s general ecological risk assessment process for HAPs.

Ecological risk assessment “evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors” (EPA 1992a). Ecological risk assessment provides a critical element for environmental decision-making by giving risk managers an approach for considering available scientific information along with the other factors they need to consider (e.g., social, legal, political, economic) in selecting a course of action.

As defined in EPA’s draft ecological risk assessment guidelines, ecological risk assessment consists of three primary phases: problem formulation, analysis, and risk characterization (EPA 1996c). This ecological risk assessment framework is shown in **Exhibit 7** and will be explained in more detail in later sections.

In the case of air toxics, ecological impacts can result from exposure to airborne HAPs (e.g., via inhalation) or exposure to HAPs deposited or transferred to other environmental media (e.g., water, soils). The HAP emissions can be assessed for both primary and secondary effects. Primary effects (e.g., lethality, reduced growth, and impaired reproduction) result from exposure of aquatic and terrestrial organisms to HAPs. Secondary effects are the result of HAP action on supporting components of the ecosystem (e.g., habitat destruction, loss of prey, and nutrient imbalances). The HAP emissions also can be assessed for both local and regional impacts. Local impacts, which apply to most HAPs, may be short-term or long-term and affect receptors near the source. Regional impacts, which apply primarily to persistent and bioaccumulative HAPs, are most often long-term and generally affect organisms both near to and distant from the source.

EXHIBIT 7  
ECOLOGICAL RISK ASSESSMENT FRAMEWORK (EPA 1996c)



Ecological risk assessments of HAPs may vary widely in scope and complexity. A screening-level risk assessment can be sufficient for some sources, while others might require a more extensive analysis. In many cases, a tiered approach can be employed in which a screening-level risk assessment with conservative exposure and effects estimates is used to distinguish between those HAPs that have the potential to cause adverse effects and those that pose negligible ecological risk. This analysis may be followed by increasingly refined assessments on those HAPs that have the potential to cause adverse ecological effects. Site-specific exposure and effects estimates may be used in order to more precisely estimate ecological risks for a given source of HAPs.

The following subsections describe the three key phases, as defined in EPA's draft guidelines, of an ecological risk assessment for air toxics. The focus is primarily on elements of the ecological assessment process that differ from the human health risk assessment methods described in Section 3.1.

### **3.2.1 Problem Formulation**

Problem formulation is a formal process for generating and evaluating preliminary hypotheses about why ecological effects have occurred, or may occur, from human activities (EPA 1996c). It provides a foundation upon which the entire ecological risk assessment depends. However, because problem formulation is inherently interactive and iterative, rather than linear, substantial re-evaluation is expected to occur within and among all products of problem formulation. Problem formulation generally involves the development of three products: assessment endpoints, a conceptual model, and an analysis plan.

#### **Assessment Endpoints**

Because an ecosystem is very complex, including many potential receptors, exposure pathways, and responses to a stressor, it is necessary to select a manageable subset of assessment endpoints when performing an ecological risk assessment. Assessment endpoints are "explicit expressions of the actual environmental value that is to be protected" or is of concern (EPA 1992a). Assessment endpoints include both a valued ecological entity (e.g., a species, ecological resource, habitat type, or community) and an attribute of that entity that is important to protect and that is potentially at risk (e.g., reproductive success, production per unit area, surface area coverage, or biodiversity) (EPA 1996c). Valued ecological entities include those without which ecosystem function would be significantly impaired, those providing critical resources (e.g., habitat, fisheries), and those perceived as valuable by humans (e.g., endangered or threatened

#### **Examples of Assessment Endpoints**

Sustained aquatic community structure, including species composition and relative abundance and trophic structure.

Sufficient rates of survival, growth, and reproduction to sustain populations of carnivores typical for the area.

Sustained fishery diversity and abundance.

Source: EPA 1997f

species). The value placed on the ecological entity may be either monetary (e.g., a fishery) or non-monetary (e.g., a recreational area).

Assessment endpoints that are ecologically relevant, are susceptible to the known or potential stressors (in this case, HAPs), represent societal values, and address management goals provide the best foundation for an effective ecological risk assessment (EPA 1996c). In order for the risk assessment to be useful, the assessment endpoints must be both ecologically relevant to the ecosystem and susceptible to the specific HAPs being evaluated. Assessment endpoints that also represent societal values and management goals are more effective in that they increase the likelihood that the risk assessment will be used in risk management decisions.

Ecologically relevant assessment endpoints represent components of an ecosystem that help sustain its natural structure, function, and biodiversity (EPA 1996c). For example, the assessment endpoints might be components of the ecosystem that contribute to the food base (i.e., primary production), provide habitat, promote regeneration of critical resources (i.e., nutrient cycling), or reflect the structure of the ecosystem (e.g., species diversity). If assessment endpoints in a risk assessment are not ecologically relevant, the results of the risk assessment might predict significant risk to the assessment endpoints selected but seriously misrepresent risk to the ecosystem of concern, which could lead to misguided risk management.

Susceptibility to the known or potential stressors (in this case, HAPs) involves both exposure and sensitivity (EPA 1996c). Sensitivity refers to how readily an ecological entity is affected by exposure to the emitted HAPs. If assessment endpoints in a risk assessment are not sensitive to the HAPs of concern, the results of the risk assessment may predict minimal risk to the assessment endpoints selected but may underestimate risk to the ecosystem of concern (assuming there are other ecologically relevant endpoints that are more sensitive).

Although many potential assessment endpoints can be identified, many are impractical (EPA 1996c). Assessment endpoints that can be measured directly are most effective. Assessment endpoints that cannot be measured directly, but can be represented by measures that are easily monitored or modeled also are useful, although some uncertainty is introduced depending on the relationship between the measure and the assessment endpoint. Assessment endpoints that cannot be linked with measurable attributes should not be selected. Measures that will be used to evaluate assessment endpoint response to exposures should be specified in the analysis plan.

### **Conceptual Models**

Potential interactions between pollutant emissions (e.g., HAP) and the assessment endpoints are explored by developing a conceptual model (EPA 1996c). The conceptual model links pollutant emissions, exposure pathways, ecological receptors, and ecological effects. The complexity of the conceptual model depends on the complexity of the problem (i.e., number of HAPs, number of assessment endpoints, nature of effects, and characteristics of the ecosystem).

Conceptual models include two principal components: risk hypotheses and a conceptual model diagram (EPA 1996c).

Risk hypotheses are statements that describe possible relationships between emissions of a pollutant, exposure, and assessment endpoint response. They include known information that sets the problem in perspective as well as the proposed relationships that need evaluation (EPA 1996c). Consequently, early conceptual models are intended to be broad in scope, identifying as many potential relationships as possible. As more information is incorporated, the plausibility of specific hypotheses is determined. The most appropriate risk hypotheses are identified for subsequent evaluation in the analysis phase of the risk assessment. The following represent examples of possible risk hypotheses for a typical ecological risk assessment (EPA 1996c).

- Nutrient loadings from septic systems, air pollution, and lawn fertilizers cause eelgrass loss in Wyquoit Bay by shading due to algal growth and direct toxicity from nitrogen.
- When a specific chemical (e.g., a HAP) is released to the environment at a specific rate, based on the chemical's  $K_{ow}$ , its mode of action, and the food web of the target ecosystem, it will bioaccumulate sufficiently in "X" years to cause developmental problems in receptors of concern (e.g., fish).

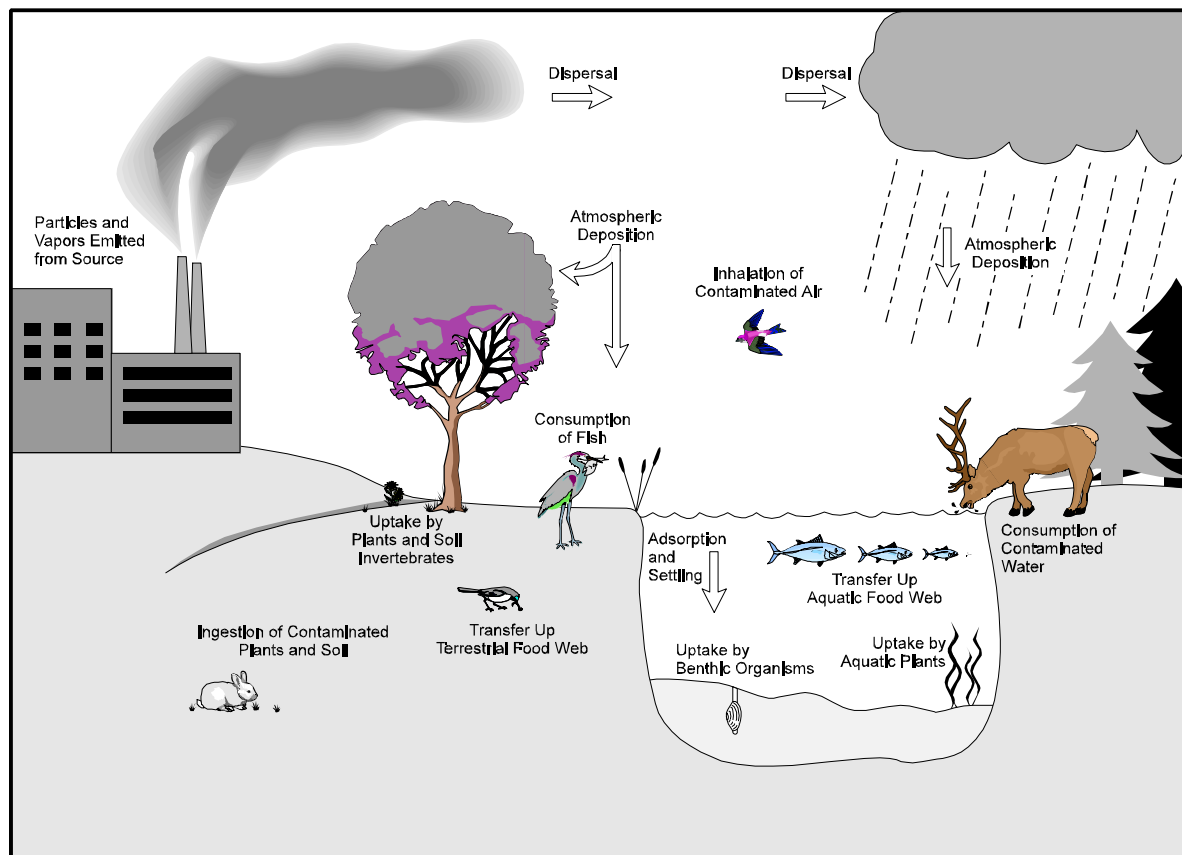
Conceptual model diagrams can communicate the relationships described by the risk hypotheses and important exposure pathways in a clear and concise way. Risk assessors can use these diagrams, along with the risk hypotheses, to select the pathways that will be evaluated in the analysis phase of the ecological risk assessment. These diagrams and hypotheses also are useful tools to aid in communication with risk managers.

The number of relationships that can be depicted in one flow diagram depends on how comprehensive each relationship is. The more comprehensive, the fewer relationships that can be shown with clarity. There is no set configuration for conceptual model diagrams. **Exhibit 8** is a sample conceptual model diagram for multipathway exposure to air toxics in aquatic and terrestrial ecosystems that was developed using a pictorial approach. **Exhibit 9** is a conceptual model diagram for exposure of piscivorous birds to HAPs that was developed using a flow diagram approach.

### **Analysis Plan**

In an analysis plan, risk assessors describe the data and measures that will be used to evaluate the risk hypotheses (EPA 1996c). Measures are identified for exposure, ecosystem and receptor characteristics, and effects. Measures of exposure quantify exposure to HAPs based on information such as source locations, emission rates, dispersion, persistence, and partitioning

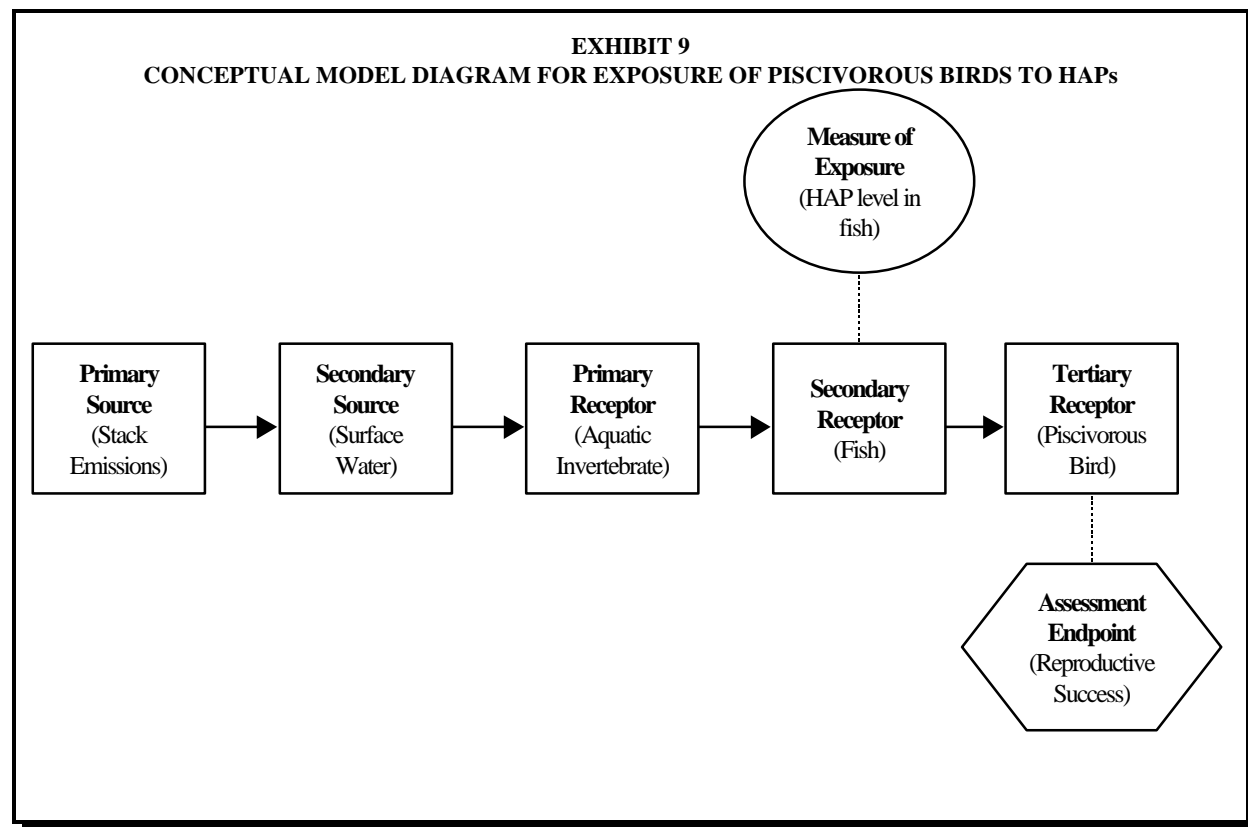
**EXHIBIT 8**  
**CONCEPTUAL MODEL DIAGRAM FOR MULTIPATHWAY ECOLOGICAL EXPOSURE TO AIR TOXICS**



properties. Measures of ecosystem and receptor characteristics identify important life history characteristics that affect the exposure or response of assessment endpoints to the HAPs (e.g., reproductive cycles, migration patterns, and habitat types). Measures of effect quantify the response of the assessment endpoints to HAP exposure (e.g., survival, growth, reproduction, and community structure).

The analysis plan also specifies how risks will be characterized. Generally, there are two ways to quantitatively estimate risks – point estimates and probabilistic estimates – and each has its advantages and disadvantages.

The point estimate approach, which has been used in numerous EPA ecological risk assessments, uses single values (usually-upper bound estimates) to represent key variables in the assessment (Finley and Paustenbach 1994). The approach is relatively simple and



straightforward; however, there are several major limitations. The repeated use of upper-bound point estimates can lead to unrealistically conservative risk estimates. In addition, point estimates provide a limited amount of information to the risk manager and the public. Therefore, the point estimate approach is most useful as a screening approach that approximates an unlikely, yet plausible, worst-case situation for some potentially exposed receptors.

In contrast, the probabilistic approach uses a distribution of data rather than a single point to represent key variables in the assessment (Finley and Paustenbach 1994). This method makes much greater use of the available exposure and toxicity data than the point estimate approach and provides more information to the risk manager. Instead of yielding a single point estimate of risk, the probabilistic approach provides a range of potential risks as well as their likelihood of occurrence. In addition, a probabilistic assessment is more conducive to sensitivity and quantitative uncertainty analysis. Major disadvantages of probabilistic assessments are that they require more time and resources and are more difficult to communicate or “sell” to some stakeholders. Another difficulty is that information on the distribution of input values is often lacking or uncertain.

### **3.2.2 Analysis**

The analysis phase, which follows problem formulation, includes two principal activities: characterization of exposure and characterization of ecological effects. The objective of this phase is to ensure that the information needed for the risk characterization phase is collected and evaluated, and interaction between scientists conducting the ecological effects evaluations and those conducting the exposure evaluations is recommended. Both activities include an evaluation of available data for scientific credibility and relevance to the assessment endpoints and the conceptual model. The products of analysis are summary profiles that describe the potential exposure to the emitted HAPs and the potential effects that may result from that exposure.

A screening-level multipathway assessment is used to identify potentially significant exposure pathways and to develop an exposure profile for ecological receptors of concern. The exposure profile is compared with: (1) published background concentrations in media and biota and (2) the levels estimated to cause adverse effects on the assessment endpoints, as described below.

#### **Characterization of Exposure**

In the exposure characterization, risk assessors describe the sources of HAPs, the distribution of HAPs in the environment, and the contact of HAPs with ecological receptors. The characterization is based on measures of exposure and of ecosystem and receptor characteristics developed in the problem formulation phase. Many aspects of the exposure characterization process, especially analyzing the sources and distribution of HAPs in the environment, are similar to the human health exposure assessment (see Section 3.1.3). The primary difference is that the exposure point for ecological receptors can differ from those for humans. Moreover, for ecosystems, exposure “areas” are usually more meaningful than exposure “points.”

#### **Characterization of Ecological Effects**

In ecological effects characterization, risk assessors evaluate the relationship between HAP exposure and adverse effects on the ecological assessment endpoints which might have been identified at the population, community, or ecosystem level. A variety of sources of ecological effects data can be used, such as field studies, laboratory studies, and structure-activity relationships.

The ecological effects characterization identifies causal information linking exposure to the HAP with relevant observed ecological effects and determines the nature and intensity of the effects and, if appropriate, the time scale for recovery after exposure ceases. The effects estimates can be either point estimates of a specified effect level (e.g., a 20 percent response level) or probabilistic estimates describing the entire stressor-response curve.

### **3.2.3 Risk Characterization**

Risk characterization is the final phase of an ecological risk assessment in which risks are estimated by integrating the estimates of exposure and effects developed in the analysis phase. As described in EPA's (1996c) proposed guidelines (and implied in the residual risk decision framework described in Section 5.4), this process requires comparison of the exposure and stressor-response profiles developed during the analysis. The ecological risk can be estimated using several approaches. One approach is to compare a single point estimate of exposure and a single point estimate of ecological effect. Another approach is to compare a distribution of exposure estimates to a single benchmark. It sometimes is possible to compare a distribution of exposure estimates to a stressor-response curve.

The risk characterization phase also should include a summary of the strengths, limitations, assumptions, and major uncertainties associated with the risk estimates. Uncertainty in risk assessment is discussed further in Section 4.2.3.

## **3.3 Data Needs for Risk Assessment**

### **3.3.1 Human Health Risk**

#### **Information Needs for Hazard Identification and Dose-response Assessment**

Regardless of the endpoint of interest (acute, chronic non-cancer, or cancer effects), consensus toxicity criteria are preferred for conducting risk assessments. For chronic non-cancer and cancer criteria, the preferred source of data is EPA's Integrated Risk Information System (IRIS). This database provides toxicity criteria that have undergone internal peer review, and, for recent assessments, external peer review, and have been approved Agency-wide. The toxicological basis for the criterion is provided, as well as other supporting data and information regarding the uncertainty in the assessment. Other chronic consensus toxicity criteria that have undergone less rigorous internal Agency review are available in HEAST, the Health Effects Assessment Summary Tables, which will be consulted for residual risk assessments when data are unavailable in IRIS. For HAPs not having adequate toxicity information in IRIS or HEAST, EPA will develop and follow a hierarchy of data sources, including various kinds of Agency health effects assessment documents, ATSDR toxicity profiles, and other sources. Consensus toxicity values for effects of acute exposures have been developed by several different organizations, and EPA is beginning to develop such values. The EPA also intends to develop and use a data source hierarchy for acute toxicity information.

**Data Needed for Chronic Non-cancer Effects Assessment.** The ultimate goal of the hazard identification and dose-response assessment for chronic non-cancer effects is to develop an RfC for inhalation exposure or RfD for oral exposure. If a consensus toxicity criterion is available from IRIS or HEAST, this value can be used in a risk assessment. If no consensus criterion is available, the risk assessor may develop a provisional RfC or RfD. The minimum database for the

development of an RfC is one well-conducted subchronic study that evaluated the respiratory tract and identified a NOAEL. A more complete database, including data from chronic studies in multiple species, and developmental and multigeneration reproductive studies, results in higher confidence in the provisional RfC. Information on a chemical's pharmacokinetics can also refine the development of an RfC. Data that the studies should provide include characterization of exposure conditions (exposure duration and concentration, and particle size distribution for particles and aerosols), endpoints examined, number of animals per group tested, and a complete reporting of the observed effects. The data needs for the development of a provisional RfD are similar. At least a subchronic oral study is needed, and the oral exposure should be adequately documented and quantified; additional data improve the assessment. These data allow the determination of whether the effect is causally associated with the chemical of interest (hazard identification), and the dose at which a significant effect occurs (dose-response assessment).

**Data Needed for Acute Non-cancer Effects Assessment.** The methodology for assessment of the risks of acute exposure is less developed than the methodology for the risks of chronic exposures. Risk assessment for acute inhalation exposure is complicated by the steep concentration-response curves that are often observed, and because small differences in exposure duration (in some cases, a few minutes) need to be taken into account. As for the chronic criteria, consensus values are preferred when available.

EPA efforts are underway to develop acute toxicity criteria with a consistent and sound scientific basis, including the acute reference exposures (AREs) being developed by ORD (EPA 1994c). Where available, AREs are the preferred values to be used for residual risk assessments. Acute exposure guidance levels (AEGLs) are being developed by an interagency group following NRC guidelines (NRC 1993b), and proposed values for the first 12 chemicals have been published for public comment (EPA 1997h). Acute toxicity criteria known as Emergency Response Planning Guidelines (ERPGs) have been developed by the American Industrial Hygiene Association (AIHA) for various severities of effects. The EPA has developed LOCs (levels of concern) for extremely hazardous substances (EHSs) regulated under section 302 of EPCRA. These and selected other acute toxicity criteria are summarized in **Exhibit 10**. Because increased exposure duration increases the incidence and severity of response, consensus acute toxicity criteria are developed for a specified duration (e.g., 1 hour). If a suitable consensus value is not available, a provisional value can be derived from acute toxicity data. The acute toxicity study should provide well-characterized exposure and effect data for the exposure route of interest. Many acute toxicity studies only report on the incidence of death. It is preferred, however, to base the development of acute toxicity criteria on studies that evaluate additional endpoints, including clinical signs, clinical chemistry, and histopathology. For an inhalation criterion, the exposure duration of the study should ideally be the same as the one of interest (e.g., one hour). If significant interpolation across exposure durations is required, multiple studies are preferred to improve the quality of the interpolation.

**EXHIBIT 10**  
**EXAMPLES OF ACUTE TOXICITY CRITERIA**

<b>Agency</b>	<b>Value</b>	<b>Definition and Basis</b>
EPA/ORD	Acute Reference Exposure (ARE)	Exposure (concentration and duration of 1-24 hours) that is not likely to cause adverse effects in the general population. Based on NOAEL/LOAEL or surrogate and UFs. Exposure levels at which increased mild (adverse effects level [AEL]-1), moderate/severe (AEL-2), or frank (FEL) effects occur also considered. Method under development.
Federal Interagency Group (includes EPA)	Acute Exposure Guidance Level (AEGl)	Under development by Federal Advisory Committee Act (FACA) committee. First 12 proposed AEGLs recently published (EPA 1997h). Concentrations for 1-8 hour exposure of the general population. Levels that are expected to protect from discomfort (AEGl-1), disability (AEGl-2), or life-threatening effects or death (AEGl-3). Based on NOAEL/LOAEL or surrogate and uncertainty factors (UFs).
EPA/OPPT	Level of Concern (LOC)	Concentration that may result in serious irreversible health effects or death in the general population after exposure for a relatively short (1-hour) period. Based on 0.1 x the IDLH (immediately dangerous to life and health) or surrogates.
American Industrial Hygiene Association (AIHA)	Emergency Response Protective Guideline (ERPG)	Concentrations for exposure of the general population for durations up to 1 hour. Levels expected to protect individuals from other than mild, transient (ERPG-1), irreversible or serious (ERPG-2), or life-threatening (ERPG-3) effects. Based on weight of evidence and professional judgment.
Agency for Toxic Substances and Disease Registry (ATSDR)	Minimal Risk Level (MRL)	For inhalation or oral exposure of the general population for up to 14 days, value at which adverse health effects not expected. Derived using NOAEL/LOAEL and UFs, similar to RfCs/RfDs.
National Research Council (NRC)	Short-term Public Emergency Guidance Level (SPEGL)	Ceiling concentration for an unpredicted single exposure (1-24 hours) designed to protect the general population. Based on professional judgment.

**Data Needed for Cancer Assessment.** As in the case of chronic non-cancer assessments, if a consensus cancer criterion is available from IRIS or HEAST, this is adequate for the assessment in question. The cancer criterion may be qualitative, in the form of a classification regarding the strength of the evidence concerning a chemical's carcinogenicity. Under the 1986 cancer guidelines, this classification might be "B2, probable human carcinogen based on sufficient evidence from animal studies." Under the proposed 1996 cancer guidelines, a chemical might be classified as "likely to be a human carcinogen by any route of exposure." These classifications represent the hazard identification phase. A dose-response assessment is also needed for any quantitative risk assessment. For cancer, this is expressed as the cancer risk per unit dose, or slope factor. If a consensus cancer criterion is not available, a provisional value may be derived. In order to derive a cancer slope factor, data are needed from a well-conducted lifetime

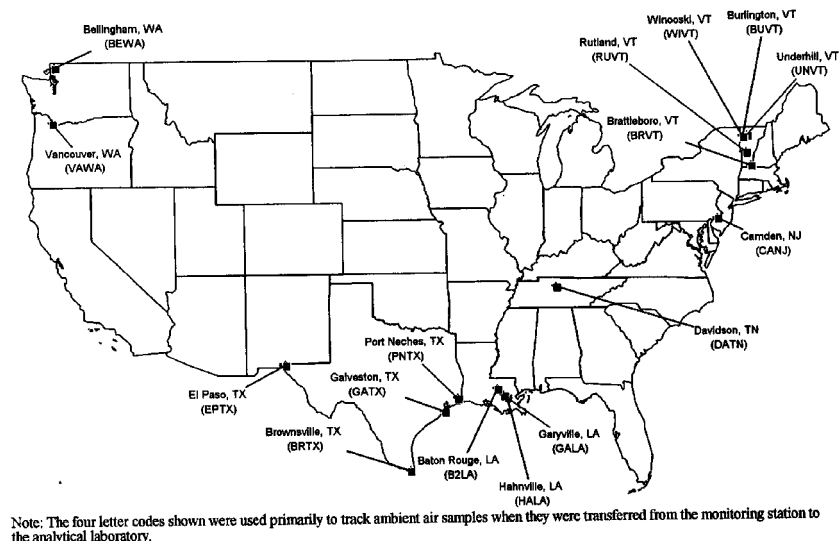
carcinogenicity study, in which an adequate number of tissues were evaluated histopathologically, and treatment-related cancer was observed. A sufficient number of animals should have been used (generally 50/sex/dose), and the incidence and type of tumor and other histopathologic lesions should have been reported. Using the cancer incidence data, a linear extrapolation to zero from a point of departure is then used to calculate the cancer risk per unit dose. Data on a chemical's pharmacokinetics, its genotoxicity, and other information on its possible mode of action can be used to refine the assessment.

### **Information Needs for Exposure Assessment**

The focus and level of detail involved in characterizing exposures depends on the scope and depth of the overall risk assessment. Ambient concentrations can be estimated for specific locations by monitoring, although the interpretation and use of monitoring data may be confounded by a number of factors. Given the widespread lack of adequate monitoring data and the difficulties with its interpretation, dispersion models are generally used in combination with emission measurements or estimates to predict ambient concentrations of hazardous air pollutants. When monitoring data are available, they can be used to check the performance of dispersion models if all sources of the HAP emissions being detected can be identified. If the monitoring network for a HAP is sufficient both temporally and geographically, and a complete source category inventory for the detected HAP is identified, monitoring data may be used to determine a "background" concentration of a HAP. Currently, the primary network of monitors available is specifically designed to assess the ambient levels of criteria air pollutants. The number of monitors for air toxics is limited, thereby limiting the usefulness for risk assessment purposes of any data that are available.

**Monitoring Data.** Presently, there is no national ambient air quality monitoring network making routine measurements of air toxics levels. Therefore, ambient data for individual HAPs are limited (both spatially and temporally) in comparison to the data available from the long-term, nationwide monitoring for the six criteria air pollutants. However, several State and local agencies operate independent toxics monitoring programs. For example, the California Air Resources Board has administered a 30-site Toxics Data Network since 1985, and the Texas Natural Resources Conservation Commission initiated a 22-site Community Air Toxics Monitoring Network in 1992. In addition, EPA sponsors several "participatory" or voluntary programs in which State and local agencies can take part through the National Volatile Organic Monitoring Contract. The "participatory" program dedicated to toxics monitoring is the Urban Air Toxics Monitoring Program (UATMP), which involves measurements of 58 volatile organic compounds and 13 carbonyl compounds. In 1995, the UATMP was comprised of 16 monitoring stations in six states (see **Exhibit 11**).

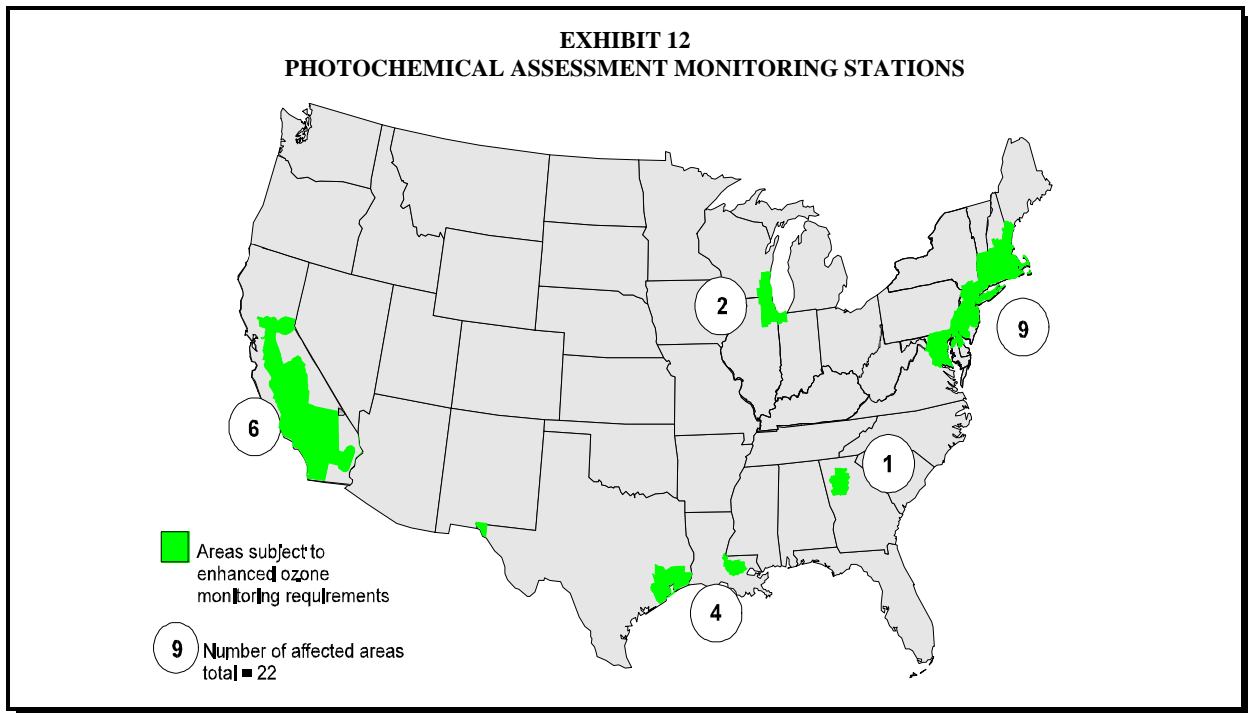
**EXHIBIT 11**  
**LOCATIONS OF 1995 URBAN AIR TOXICS MONITORING PROGRAM SITES**



Without a national mandate for ambient monitoring for toxics, there is also little incentive for the data from these various programs to be centrally archived. The EPA's Office of Air Quality Planning and Standards (OAQPS) is attempting to remedy this problem. The OAQPS is currently conducting a nationwide study to identify, catalogue, and characterize all available ambient air quality data for toxics, and to make these data publicly accessible for analysis of air toxics issues. Although a multi-year effort, the first phase was completed in the fall of 1997, and a report describing the data identified and/or collected to date will soon be published.

Although designed primarily as an effort to monitor and characterize ozone precursors, the Photochemical Assessment Monitoring Stations (PAMS) program also includes measurement of nine HAPs: acetaldehyde, benzene, ethyl benzene, formaldehyde, hexane, styrene, toluene, 2,2,4-trimethylpentane, and xylene. Initiated in February 1993, the PAMS program requires the establishment of an enhanced monitoring network in all ozone nonattainment areas classified as serious, severe, or extreme. The 22 affected areas, shown in **Exhibit 12**, cover 113 thousand square miles and have a total population of 79 million people (approximately 30 percent of U.S. population). Each PAMS network will consist of as many as five monitoring stations, depending on the area's population. Generally, each PAMS network will consist of four different monitoring sites (Types 1, 2, 3, and 4) designed to fulfill unique data collection objectives. The Type 2 sites, referred to as the maximum precursor emissions impact sites, are capable of measuring a greater array of precursors and are also particularly well-suited for the evaluation of

urban air toxics. The Type 1 site is located upwind of the metropolitan area to measure transport into the area while the Type 4 site is located downwind of the nonattainment area. The Type 3 stations are intended to measure maximum ozone concentrations and are sited downwind of the urban area. The data collected at the PAMS sites include measurements of a target list of 56 hydrocarbons and 3 carbonyl compounds on either a hourly or 3-hour basis during the ozone season as well as meteorological data. The PAMS program may play a significant role as a foundation for future ambient monitoring for air toxics.



However, for the purposes of risk assessments, specifically residual risk assessments, even comprehensive and high quality monitoring data would not be adequate and would need to be supplemented with modeling data. For example, the changes in ambient HAP concentrations over time, particularly in media such as soil, water, and biota, cannot be measured at the present time but can be estimated through modeling. Moreover, the contributions of individual sources and source categories often cannot be determined based on monitoring data alone.

**Data Needed for Emissions Modeling.** For an exposure assessment of a stationary source of HAP emissions, emissions data are needed. Ideally, the emission estimates are from direct measurements of source stack emissions. Although direct measurement is likely to provide the most accurate data for an emission source, these data are not always available, as such sampling is often time- and resource-intensive. When specific emission measurements are not feasible or available, other emission estimation methods including material balances and emission factors are sometimes used as an alternate method. Emission factors indicate the quantity of a pollutant typically released to the atmosphere for a particular source operation, and are usually

representative of an industry or emission type as a whole. Actual emissions from a specific source may be higher or lower or may be comprised of a different set of individual HAPs than the emission factors indicate because of site-specific process design, control equipment, operation and maintenance practices, or other factors. Before using an emission factor, available documentation on how the emission factor was derived should be studied to determine whether it is appropriate for the source under consideration. Each approach to estimating emissions has an inherent level of uncertainty which adds to the overall uncertainty of a risk analysis.

In addition to estimating the quantity of emissions, release characteristics of the source must be defined. Knowledge of the emission rate and release characteristics enables the pollutant fate and transport to be estimated. Modeling of emissions released from a stack requires knowledge of the stack height, inner stack diameter, gas exit velocity or flow rate, gas exit temperature, and knowledge of the nearness of structures to the release point. For small sources within a larger facility (e.g., emissions from storage piles or ponds), the dimensions of the small source should be identified. While point source emission rates are expressed in terms of mass per unit time, non-point source emission rates are more typically modeled in terms of mass per unit time per unit area. Another important consideration in specifying the source emission rates is whether the rates should reflect short-term or annual operating conditions. Ideally, it is better to have hourly or daily emission rates; however, these data are not typically available. Short-term emission rates provide the flexibility to model emissions over a range of release times, to assess risk over shorter intervals than annual, and to permit more accurate assessments through the incorporation of microenvironment and population activity pattern analyses.

After the pollutants of interest and their sources and emission rates are defined, the risk assessment process continues with estimation of the pollutant fate and transport. Initially, the diffusion of the emitted pollutants is largely determined by the source release characteristics. After pollutants are released to the atmosphere, their transport and dispersion are governed by meteorological principles, terrain characteristics, wet and dry deposition rates, and certain chemical properties of the HAP (such as aqueous solubility, vapor pressure, air-water partition coefficient [i.e., Henry's Law constant], molecular diffusivity, phase portion coefficient, melting point, and absorptivity). A variety of mathematical models have been developed to describe the transport and fate of pollutants released to the atmosphere, each with specific data needs.

**Data Needed for Population Assessment.** Exposure and risk to human populations via the inhalation route involves combining pollutant concentration information with information on the geographical distribution of people in the study area, including consideration of data on the activities and characteristics of the exposed population. Human exposure and susceptibility and sensitivity to pollutant effects may vary with factors such as age, intensity and amount of activity, time spent in microenvironments, diet, overall health, lifestyle, and the concentration of pollutant. The extent to which these factors are included in the risk assessment depends on the purpose of the assessment, available resources, uncertainties in the assessment, and data quality and quantity.

After ambient levels of air pollutants have been estimated using either dispersion modeling or monitoring data, the exposed population must be characterized. The U.S. Bureau of Census is a major source of population information. The EPA uses data that are based on the census block level. There are about 6.9 million census blocks in the U.S. The number of people residing in each census block and the geographical center of each are specifically used in the assessments. The population included within a census block is highly variable (from less than 10 to a few thousand), but, on average, about 30 to 40 people reside in each block. These data provide a good estimate of how people are geographically distributed near emitting sources, and are also useful for defining the population cohorts for analysis. Cohorts may be defined on the basis of age, gender, race, income levels, length of time in primary residence, or other characteristics. Data on population characteristics relevant to exposure potential are obtained from documents and studies such as EPA's *Exposure Factors Handbook* (EPA 1997d) and national population surveys of people's activity patterns, including where they spend each hour of a day (microenvironment) and each hour's activity level (EPA 1994h).

**Data Needed for Non-inhalation Exposure Modeling.** Various equations and scenarios are available for modeling exposures that occur through routes other than inhalation, and each equation requires the appropriate input data. The simplest screening-level multipathway exposure assessments require chemical-specific data (e.g.,  $K_{ow}$ ) to model the partitioning of the chemical in the environment and uptake rates (e.g., 3 liters water/day) to predict intakes. Combining this information yields general predictions of non-inhalation exposure.

More complex modeling of non-inhalation exposures requires a substantially greater number of input parameters. A full multimedia, multipathway exposure assessment typically accommodates surrounding terrain (e.g., nearby waterbodies) and land use (e.g., vegetable gardening). The associated equations for such an analysis typically start with atmospheric deposition rates and require additional chemical data and many other input parameters related to the environmental setting and population. For example, modeling pollutant fate and transport through a waterbody requires information such as waterbody location, size, and drainage area for each waterbody being evaluated. As another example, modeling exposure via vegetable consumption involves parameters such as soil type, soil depth, annual rainfall, and vegetable type (e.g., root, leafy).

As with inhalation, assessing non-inhalation exposure to human populations involves combining pollutant concentration information with information on the numbers, geographical distribution, and characteristics of people in the study area, including consideration of information on the activities and behavior of the exposed population. The kinds of information needed include soil, drinking water, and food ingestion rates (often including specific foods, such as fish, beef, pork, eggs, root vegetables, grains, fruit), generally for both adults and children, as well as contact frequencies with soil and surface water. Some activities of interest for non-inhalation modeling are subsistence farming and subsistence fishing because of the unique dietary habits of these two groups (i.e., eating much more garden vegetables and fish, respectively). Also, as with inhalation exposure, the extent to which these factors are included in the risk assessment depends

on the purpose of the assessment, available resources, uncertainties in the assessment, and data quality and quantity. Not only are the data requirements often extensive, particularly when many different pathways are being assessed, but the computational demands also can be quite large in a multimedia, multipathway assessment. For further information about specific data needs, see the multipathway exposure assessment methodology documents cited in Section 3.1.3.

### **3.3.2 Ecological Risk**

#### **Information Needs for Ecological Effects Characterization**

To characterize ecological effects, risk assessors need to evaluate the relationship between HAP exposure and adverse effects on the ecological assessment endpoints. As discussed in Section 3.2.2, the relationship can be represented using a point estimate of ecological effects at a specified exposure concentration (e.g., a threshold for effects) or a full stressor-response curve. Different types of original data can be used for ecological effects characterization. These include field studies, microcosm studies, laboratory studies, and structure-activity relationships. These data types are described below.

- **Field Studies.** Studies of wildlife, populations, communities, and ecosystems exposed to HAPs in natural settings can provide valuable information on the effects of HAPs. In many cases, however, wildlife are exposed to numerous types of stressors (chemical and non-chemical), and the effects of individual HAPs can be difficult to isolate. In addition, field studies are conducted infrequently due to the time and resources required.
- **Microcosm Studies.** Studies on the exposure of multi-species and multi-media enclosed experimental systems to HAPs can control some of the uncertainty associated with multiple stressor exposure in field studies. These studies can provide information about food web dynamics and the interactions of populations of organisms. As with field studies, microcosm studies are time and resource intensive and, therefore, are relatively uncommon.
- **Laboratory Studies.** Due to the limitations and expense of field studies and microcosm studies, most risk assessors rely on laboratory toxicology studies. These studies are typically easier to conduct, and effects can be directly linked to exposure to a single HAP. There is uncertainty, however, in extrapolating the results from standard laboratory species to the wide array of wildlife species. Additionally, in most cases, laboratory studies are not designed to assess effects on populations, communities, and ecosystems.
- **Structure-Activity Relationships (SARs).** In the absence of adequate laboratory studies, scientists may rely on SARs. By using SARs, the toxic effects of a HAP can be inferred based on the similarity of its chemical structure to a chemical with known toxic effects. Types of SARs include: quantitative SARs (QSARs), qualitative SARs, and best analog SARs.

### **Information Needs for Ecological Exposure Characterization**

In order to characterize exposure to a pollutant or stressor (e.g., HAPs), risk assessors need to describe its sources, its distribution in the environment, and its contact with ecological receptors. Much of the information used in this characterization is similar to that used for the human health exposure assessment (see Section 3.3.1). For example, monitoring data and emissions modeling are major sources of information regarding the sources of pollutants (e.g., HAPs) and their distribution in the environment.

In addition, multimedia exposure to pollutants of concern and the potential for their subsequent bioconcentration and biomagnification in aquatic and terrestrial food webs often are important for both human health and ecological risk assessment. Consequently, risk assessors need fate and transport data specific to the pollutant of concern, such as a pollutant's octanol-water partition coefficient ( $K_{ow}$ ), organic carbon-water partition coefficient ( $K_{oc}$ ), and bioconcentration factor (BCF) or bioaccumulation factor (BAF) values.

However, because ecosystem characteristics are site-specific, some of the information needed to characterize the contact of a pollutant such as a HAP with potential receptors are specific to the ecological risk assessment methodology. For example, an understanding of the site characteristics, including such factors as site topography, soil and water types, and habitat types, is important. Furthermore, the "significance" of potential ecological effects depends on other site-related factors, including the type and significance of the ecological receptors affected and the areal extent of exposures at concentrations sufficient to cause adverse effects. Tools risk assessors can use to determine the locations and types of ecological receptors in areas surrounding the sources include information gathered using maps (e.g., U.S. Geological Survey, National Wetlands Inventory, and EPA's ESTAT Geographical Information System), aerial photographs, communication with scientists knowledgeable about the area (e.g., U.S. Fish and Wildlife Service, National Oceanic and Atmospheric Administration), and site surveys.

In the absence of readily available site-specific information and prior to the recommendation of a site specific ecological risk assessment, the risk assessor can use approximate source location information to infer the existence of adjacent aquatic and terrestrial ecosystems and a set of assessment endpoints can be selected that represent the most appropriate sensitive elements of those ecosystems for the contaminants in question. OAQPS currently is testing such an approach in screening HAPs for ecological risks.

### **3.4 Mixtures Assessments**

The EPA mixtures guidelines (EPA 1986d), which are currently in the process of being updated (EPA 1997g), indicate the following hierarchy for evaluating mixtures:

- Use toxicity data on the specific mixture of concern;
- If such data are not available, use toxicity information on a similar mixture; and
- If such data are not available, use toxicity information on the components of the mixture.

It is unlikely that mixtures of HAPs from sources under review for residual risk will have been studied as independent entities because of their variability. Thus, the default has been and will continue to be to evaluate data on the individual mixture components, in accordance with EPA's guidelines.

#### **3.4.1 Non-cancer Effects**

For non-cancer assessment, the default approach, in the absence of information about interactions between components of the mixture, is to assume additivity. This simple approach will generally be used for screening-level residual risk assessments. The assumption of additivity is inherent in the use of the HI for evaluation of non-cancer risks. If the HI ( $HI = \sum HQ = \sum \text{exposure/health criterion for all mixture components}$ ) exceeds

The hazard quotient (HQ) is the ratio of the estimated exposure (EXP) to the health criterion level for a given chemical. For example, for chronic exposure, the health criterion could be the RfC. If the HQ,  $EXP/RfC$ , is greater than 1, the RfC is exceeded and a potential health risk is present. If the HQ is less than 1, the RfC is not exceeded and health effects are unlikely. The hazard index (HI) is the sum of the HQs for each chemical in a mixture. If the HI is greater than 1, a potential health risk is present; if  $<1$ , health effects are unlikely.

1, an unacceptable health risk is possible. The 1986 guidelines go on to state "Since the assumption of dose addition is most properly applied to compounds that induce the same effect by similar modes of action, a separate HI should be generated for each endpoint of concern. Dose addition for dissimilar endpoints does not have strong scientific support...". This guidance has been translated in practice in the *Risk Assessment Guidance for Superfund* (EPA 1989c), which indicates that "it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group". Further guidance from this document also is relevant here: "Segregation of hazard indices by effect and mechanism of action can be complex and time-consuming because it is necessary to identify all of the major effects and target organs for each chemical and then to classify the chemicals according to target organ(s) or mechanism of action. This analysis is not simple and should be performed by a toxicologist. If the segregation is not carefully done, an underestimate of true hazard could result. Agency review of particularly complex or controversial cases can be requested..." (EPA 1989c).

In practice, specific hazard indices often are determined on the basis of the target organ, since there are not usually adequate data available to distinguish chemicals based on mode of

action. Such a target organ approach, which is conservative, generally will be taken in the refined residual risk assessment. In theory it is possible to distinguish different modes of action, even when the effect is in the same tissue, but in practice there is generally inadequate information to do so.

The issue that arises in developing target-specific hazard indices is the availability of health criteria for various effects or target organs. The RfC is based on one or a few effects that occur at the lowest concentration, but other target organs may be affected at higher concentrations. The revision of the mixtures guidelines currently under development will discuss this issue in detail, and the current draft recommends derivation of target tissue effective concentrations, essentially analogous to developing RfCs for each affected endpoint. At present, however, health criteria values are not available for less sensitive effects. Again, the *Risk Assessment Guidance for Superfund* (EPA 1989c) offers guidance for this situation: "Although higher exposure levels may be required to produce adverse health effects other than the critical effect, the RfD can be used as the toxicity value for each effect category as a conservative and simplifying step." According to this guidance, an appropriate HI would be calculated for all components of a mixture that affect the same target organ using the RfC (even if the RfC was derived based on an effect in a different target organ).

### **3.4.2 Cancer**

For carcinogens with the assumption of linear dose response curves, the estimated increase in risk at an estimated exposure level can be summed across all components of a mixture. This assumption of risk additivity of linear carcinogens is appropriate for both the screening-level and refined residual risk assessments.

A variation of the additivity approach is used for some mixtures of structurally similar carcinogens for which cancer slope factors (i.e., potency) are not available for all mixture components. For carcinogenic dioxins and furans, for example, a toxic equivalency factor (TEF) approach is used, as described in EPA's dioxin reassessment document (EPA 1994i). In this approach, which has an underlying assumption of additivity across mixture components, the cancer potency of certain dioxin and furan congeners is estimated relative to 2,3,7,8-TCDD based on other toxicity information that is available for all the congeners (e.g., LD<sub>50</sub>). Then, TEFs based on these relative cancer potencies are used to adjust the exposure concentrations of mixture components, which are subsequently summed into a single exposure concentration for the mixture. That exposure concentration based on TEFs is then used, along with the 2,3,7,8-TCDD slope factor, to estimate cancer risks for the mixture.

For carcinogens with risk assessment based on the assumption of nonlinear dose response, the proposed MOE approach may be used in the refined assessment, consistent with the proposed revision of EPA's cancer guidelines (EPA 1996b). Since neither thresholds nor risk are explicitly estimated, there is no analogous form of the simple dose addition approach that is amenable. Since the MOE analysis is done on a case by case basis, the determination of the appropriate

“acceptable” MOE (see Section 4.1.1) for each component would be required before a mixtures assessment could be performed. Using the “acceptable” MOE so derived, a health criterion could be derived as the point of departure divided by the “acceptable” MOE, which is determined by the risk manager after being presented with a complete cancer risk assessment. This level would not be interpreted as a zero-risk dose, but as a dose considered to be low enough to be appropriate under the specific program constraints. Given this value, an HI approach could be applied using the estimated exposure level for each mixture component and the health criterion derived in this way for each component, since an assumption of additivity would likely be conservative for nonlinear dose response curves. Assuming additivity of nonlinear carcinogens is assumed to be conservative for the same reason that additivity of non-cancer health criterion values is conservative, because general additivity would include addition of effects that occur in different target tissues or by different mechanisms of action. There remains the possibility of potentiation or synergistic interactions, but these cannot be predicted, and are not likely to be prevalent. In general, the assumption of additivity is expected to be a conservative one.

The MOE approach leaves the decision about the appropriate reduction in exposure compared to the point of departure (i.e., the observable toxicity data) up to the risk manager. An in-depth MOE analysis would be made in consideration of factors that could include the steepness of the dose-response curve, persistence of the compound in the body, known human variability in response, or demonstrated human sensitivity as compared with experimental animals. An alternative to this approach that may be useful as a screening tool is to apply a default “acceptable” MOE value to all carcinogens and then to assume additivity, as described in more detail in Section 4.1.1. A default “acceptable” MOE of 1,000 could be used as a screening tool, for example. In a typical case, the point of departure derived from modeling the observable data would be a tumor incidence of 10 percent (e.g., risk of 1 in 10). If the chemical fits a linear mode of action, a reduction in the dose of 1,000 would result in an estimated risk of 1 in 10,000. For a nonlinear mode of action, a reduction of the same magnitude would lead to a much lower risk (possibly to zero risk) because of the nonlinearity in the dose-response slope. It is reasonable to assume that the nonlinearity in a cancer-related mode of action would be evident at dose levels near the observable data range, since such mechanisms often are related to a non-cancer effect that has some enhancing influence on the development of tumors. Therefore, the aspects of a nonlinear carcinogenic mode of action that result in a more rapid reduction in cancer risk with reduction in dose can be assumed to be relatively more active at doses just below the observable range. Although the magnitude of the nonlinearity cannot be known *a priori*, the adoption of a relatively large MOE ensures conservatism and can be used as a screening tool.

It is also not clear how the MOE approach should handle effects in different target organs or with different modes of action. A consideration of the mode of action that leads to the conclusion that the nonlinear dose-response evaluation is appropriate can also provide information relevant to whether nonlinear carcinogens should be considered additive. In general terms, additivity should be assumed unless contrary information is available. Carcinogenic responses arising in the same tissues should be considered additive, unless detailed mechanistic information is available showing the mechanisms are unrelated. Carcinogenic substances showing nonlinear

modes of action through unrelated mechanisms or in different tissues would not generally be combined.

### **3.4.3 Ecological Effects**

As for the case for non-cancer assessments of human health risks, when ecological toxicity data for complex mixtures are unavailable, the HI approach can be used to integrate the ecological risks of multiple chemical stressors (EPA 1996c). Quotients for the individual constituents in a mixture are derived by dividing each constituent's exposure level by a corresponding benchmark for ecological effects (see Section 5.4). The resulting quotients are then added together to generate an overall HI for the mixture. If the HI of the mixture is greater than 1, a risk to ecological receptors is assumed.

The HI approach assumes that the toxicities of the mixture constituents are additive or close to additive. This assumption is likely to be true for mixtures of chemicals that have similar modes of action. For mixtures of chemicals that have dissimilar modes of action, additivity or less-than-strict additivity was shown to be common based on fish acute toxicity tests (EPA 1996c). However, the ecological toxicity of a chemical mixture may be greater (synergistic) or less (antagonistic) than what is predicted by considering the toxicities of the individual chemicals in the mixture to be additive. Thus, the residual ecological risk assessment can use the HI approach to estimate the risks of mixtures of HAPs to ecological receptors, but the assumptions and associated limitations concerning HAPs interactions should be clearly stated in the assessment's documentation. It may often be the case that a single chemical is responsible for the HI exceeding 1, and the assessment can focus on that chemical.

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## **4. Other Statutory Report Requirements**

The preceding chapter describes the methods available for performing human and ecological risk assessment and provides an overview of the risk assessment process that will be used to evaluate residual risks for HAPs. The specific strategy that the Agency is currently evaluating for use in the residual risk program is described in Chapter 5. The remaining elements required by statute to be covered in the section 112(f)(1) Report to Congress are addressed in this chapter. Additional aspects of some of these topics are also covered in other parts of this report.

### **4.1 Section 112 (f)(1)(B)**

Section 112(f)(1)(B) of the Clean Air Act directs EPA to investigate and report on “...public health significance of such estimated remaining risk and the technologically and commercially available methods and costs of reducing such risks.” These topics are presented in the following two sections.

#### **4.1.1 Public Health Significance**

This section addresses the directive in CAA section 112(f)(1)(B) that EPA investigate and report on “...public health significance of such estimated remaining risk...” Currently the data are not available to conduct an analysis to determine the public health significance for air toxics. In addition, EPA has not completed any residual risk analyses for specific source categories.

EPA is proposing to determine the public health significance associated with the residual risk program according to a decision framework that is described below. Risk that is judged to be significant, under the framework, would be subject to regulation. This section first discusses EPA's general framework for determining the public health significance of risks remaining after application of a MACT standard to a source category (i.e., determining whether or not residual health risks need to be reduced), and then discusses the health-related criteria EPA intends to use to evaluate public health significance in the residual risk program.

Given the legislatively mandated schedules for MACT implementation and for performing residual risk assessments, risk analyses have not been completed by EPA on any source categories for the purposes of estimating potential residual risks. Without these analyses, there is no way to determine currently what the public health significance of any identified residual risks may be. As residual risk assessments are completed for individual source categories, EPA will evaluate public health significance by applying the ample margin of safety framework as part of its decision-making process.

### **EPA's General Framework for Evaluating Public Health Significance**

The EPA believes the ample margin of safety concept as introduced in the 1970 CAA Amendments, and as applied in the benzene standard (EPA 1989b), remains valid and is a reasonable approach to evaluate public health significance and to manage residual risks under CAA section 112. Such an approach is consistent with the Congressional language in section 112(f)(2) (see Appendix A). While no guidance on how to apply ample margin of safety was available from the 1970s through the late 1980s, the 1989 benzene NESHAP presented a structure for applying ample margin of safety to carcinogens. The following paragraphs describe how EPA applied the ample margin of safety framework to benzene.

EPA developed the benzene risk management framework in response to a 1987 D.C. Circuit Court decision on the *Vinyl Chloride* national emission standard, also taking into consideration public comment on several alternative risk management approaches it had proposed for benzene (see Section 2.1 for more historical background on the benzene national emission standard). According to the benzene framework, EPA would develop national emission standards for HAPs in two steps: (1) first determine a “safe” or “acceptable risk” level, considering only public health factors, and (2) then set an emission standard that provides an “ample margin of safety” considering relevant factors in addition to health such as costs, economic impacts, and feasibility. In establishing the acceptable risk level, EPA would consider the extent of the estimated risk were an individual exposed to the maximum level of a pollutant for a lifetime, i.e., maximum individual risk (MIR). Although an MIR for cancer of approximately 1 in 10 thousand should ordinarily be the upper end of the range of acceptability under this approach, EPA would consider other health and risk factors (e.g., projected overall incidence of cancer or other serious health effects within the exposed population, the number of people exposed within each individual lifetime risk range, the science policy assumptions and estimation uncertainties associated with the risk measures). In the second step, EPA would attempt to provide protection to the greatest number of people possible at an excess individual lifetime risk of cancer no higher than 1 in 1 million ( $10^{-6}$ ), taking into account additional factors relating to the appropriate level of control (e.g., costs, economic impacts, feasibility). The acceptable risk established in the first step would not be exceeded by the standards EPA adopts based on the second step. This approach is consistent with risk management approaches taken by other EPA programs intended to protect public health. For example, RCRA and CERCLA programs use a risk management range of  $10^{-6}$  to  $10^{-4}$  under their reasonable maximum exposure scenario to guide their decision-making for carcinogens.

The EPA has not yet implemented the ample margin of safety approach as interpreted by the *Vinyl Chloride* decision with respect to non-cancer and carcinogens for which the MOE analysis is appropriate though the EPA believes that the 1989 benzene NESHAP could provide important guidance for residual risk decisions in this areas. In applying the benzene NESHAP approach, the EPA would first determine an “acceptable” level of such risk, again without taking into consideration the cost of achieving such protection or other, non-health factors. As a second

### **MIR, MEI, and Individual Most Exposed**

Maximum individual risk (MIR) is a concept included in the benzene decision and is similar but not identical to the concept of maximum exposed individual (MEI). An MIR represents the highest estimated risk to an exposed individual in areas that people are believed to occupy. The MEI represents the highest estimated risk to an exposed individual, regardless of whether people are expected to occupy that area. Thus,  $MEI\ risk \geq MIR$ .

In the residual risk program, the MEI risk estimate will be used in screening residual risk assessments but not in refined assessments, whereas the MIR estimate will be used in refined residual risk assessments and may be used in screening assessments. Because screening-level risk assessments will be employed for the purpose of determining whether or not further analysis and concern are warranted, the MEI estimate may be used for risk management decisions that result in the judgment not to regulate a given source category, but will not be used for risk management decisions that call for additional controls or regulatory actions.

The EPA is proposing that the "individual most exposed," a phrase used in CAA section 112(f)(2), be considered equivalent to the MIR for the purposes of regulation under the residual risk program.

step, EPA would set standards sufficient to provide an "ample margin of safety" in which these other factors would be weighed. Should EPA decide to proceed in this direction, the Agency would have the discretion under *Vinyl Chloride* to identify both the "acceptable risk" level and methods of arraying factors for consideration in the "ample margin of safety" step.

The Agency could choose to follow an alternative framework for such risks as long as it complies with *Vinyl Chloride*, including, for example, a one-step framework which proceeds directly to an "ample margin of safety." EPA believes that, under *Vinyl Chloride*, any such one-step framework would not be permitted to take into consideration costs and other non-health factors. A framework to make air management decisions for non-carcinogens and carcinogens for which an MOE analysis is appropriate is under development and will not be available in time to include in this Report. A summary of where EPA will use the benzene framework is given in **Exhibit 13**.

### **EXHIBIT 13 APPLICABILITY OF THE BENZENE NESHAP FRAMEWORK**

<b>Nature of HAP Hazard</b>	<b>Residual Risk Framework</b>
Carcinogens for which an MOE analysis is appropriate	To be determined
All other carcinogens	Benzene NESHAP two-step framework
Non-carcinogens	To be determined

Section 112(f) also gives EPA the authority to promulgate more stringent controls necessary to protect against an adverse environmental effect. In promulgating such controls, EPA

must take into consideration costs, energy, safety and other relevant factors. The EPA is currently developing a policy for how it will implement this authority.

### **Health-related Criteria to Be Used in Evaluating Public Health Significance**

The EPA will apply the ample margin of safety framework to public health risks in the context of the tiered, iterative risk assessment and management approach for air toxics' residual risks. For carcinogens, EPA will apply a two-step ample margin of safety approach, as described above and in Section 2.1. It will use somewhat different health-related criteria to evaluate the public health significance (i.e., ample margin of safety) of screening-level risk assessment results and refined risk assessment results. This distinction is appropriate given the greater degree of conservatism built into the screening-level assessments, and EPA's strong desire to minimize false negative outcomes based on screening analyses. These health-related criteria for carcinogens are described below and summarized in **Exhibit 14**. Some of the criteria EPA believes are important in assessing the public health significance (i.e., ample margin of safety) and thus likely to be part of any future approach are discussed below, in more general terms than the criteria for benzene.

**EXHIBIT 14**  
**SUMMARY OF CRITERIA FOR EVALUATING PUBLIC HEALTH SIGNIFICANCE**  
**FOR CARCINOGENS**

<b>Effect Type</b>	<b>Screening Level<sup>1</sup></b>	<b>Refined<sup>2</sup></b>
Cancer	<ul style="list-style-type: none"><li>▶ Upper-end individual risk <math>&lt;10^{-6}</math> generally meets ample margin of safety; <math>\geq 10^{-6}</math> generally leads to refined analysis</li><li>▶ Assume additivity for all HAPs</li><li>▶ Confidence in toxicity values not necessarily considered</li><li>▶ Size and nature of potentially exposed population not necessarily considered</li></ul>	<ul style="list-style-type: none"><li>▶ Upper-end individual risk <math>&lt;10^{-6}</math> generally meets ample margin of safety; <math>\geq 10^{-4}</math> generally does not meet ample margin of safety</li><li>▶ Upper-end individual risk between <math>10^{-6}</math> and <math>10^{-4}</math> may meet ample margin of safety, depending on confidence in the risk estimate, population size, presence of sensitive subpopulations at various risk levels, and other factors</li><li>▶ Assume additivity for all HAPs</li></ul>

<sup>1</sup> Screening based on upper-end estimated HAP exposure at the location of either the MIR or MEI. All available toxicity values will be considered.

<sup>2</sup> Refined assessment based on more detailed and site-specific, and less conservative, estimated HAP exposures at the MIR location and throughout the spatial area of impact. EPA consensus toxicity values, or equivalent, are required.

The screening-level risk characterization will be used by EPA managers to decide if a more refined risk assessment should be conducted (i.e., ample margin of safety not met based on screening results), or if nothing more needs to be done under the residual risk program (i.e., ample margin of safety exists). This decision will not be made without first discussing the results with the involved stakeholders. EPA will make information available to any State or local air toxics agency, the industries affected, and any concerned public interest groups.

**Criteria for Evaluating Screening Analysis Results.** EPA will consider a wide range of available toxicity values in determining if the continued emission of HAPs poses a risk to the public or the environment. When EPA-verified toxicity values are not available, other sources of toxicity values may be used (see Section 3.3.1). The use of other toxicity values in screening analyses expands the database available for each HAP and provides more information on which to base decisions.

Cancer. The assessment of cancer risks will follow the direction of the 1996 proposed cancer guidelines, which utilize information on the mechanism of action more than the previous guidelines. A linear mechanism is assumed for screening, and the additive individual lifetime cancer risk for all HAPs combined should be less than approximately  $10^{-6}$  at the location of either the MIR or MEI. Otherwise, a more refined risk analysis generally is needed. The  $10^{-6}$  level of concern for the initial screen is consistent with the benzene NESHAP and follows CAA section 112(f) requirements that facilities that show a lifetime excess individual risk of cancer greater than one in a million should be considered for additional regulations. As noted above, for purposes of the residual risk program, excess individual lifetime cancer risk of approximately  $10^{-6}$  will be considered a trigger for more refined analysis. Where the screening risk results are below this level, an ample margin of safety determination (considering risk and other factors) will be made to confirm that an ample margin of safety exists. If so, no further risk analysis is needed, and no further risk reduction will be required under section 112(f). If risks are not within an ample margin of safety, additional analysis is necessary.

Non-Carcinogens. At the screening stage, the health criterion for all non-cancer assessments (acute and chronic) will be based on the hazard index (HI) calculated by assuming additivity of HAPs in a mixture. For each HAP emitted from a source category's facilities, the toxicity value will be compared with the upper-end HAP exposure level, as determined in the exposure screen, resulting in a hazard quotient (upper-end HAP exposure level  $\div$  toxicity value [such as the RfC]). The hazard quotients for each HAP in the mixture will be added regardless of endpoints, resulting in an HI value. This will result in a more conservative outcome than looking at HAPs individually, or than looking at different endpoints separately. While EPA's risk management policy for non-cancer effects is not yet available, a more refined risk assessment would likely be conducted when the HI exceeds 1 in the screening analysis (i.e., when exposure estimates exceed toxicity reference levels). At the screening stage, there would be no in-depth consideration of the size of the UF(s) used in calculating individual toxicity values or in the confidence in those values. Acute and chronic exposures will be assessed separately. For chronic exposures, long-term exposure estimates (e.g., annual average) will be used. For acute risks, a

similar analysis will occur except that short-term exposure estimates (e.g., one-hour averages) will be used.

**Criteria for Evaluating Refined Analysis Results.** The refined analysis, which can itself be iterative and include several tiers of increasing complexity, will be used to determine whether HAP emissions from a source category pose a continued risk to the public or the environment and whether additional emission reductions are needed. The refined risk assessment increases the level of confidence by requiring that EPA consensus toxicity values, or equivalent, be used. This requirement ensures that toxicity criteria of consistently high quality and derived by a consistent methodology are used in the assessment. Any toxicity value used in a refined risk assessment other than an EPA consensus value will be subjected to an Agency peer review process. The refined analysis also differs from the screening analysis in that the approaches to addressing mixtures are more detailed. In the exposure assessment, more site-specific data and more refined models are used to estimate exposure concentrations and intakes. In addition, the refined analysis considers the number of people exposed at various levels along with the MIR; the MEI risk estimate is not considered in the refined analysis.

Carcinogens Subject to Benzene NESHAP. In assessing the cancer risk in the refined analysis, multiple HAP exposures are treated as additive regardless of target organ, and the number of people exposed in various subpopulation groups are considered. The intent is to provide some way of fine tuning the assessment so that specific populations may be protected to a greater degree, when appropriate. As stated above and in section 2.1, as a general policy, no individual risk greater than approximately  $10^{-4}$  should exist for any member of the general population. This is consistent with the benzene NESHAP, which states that “an MIR of approximately [ $10^{-4}$ ] should ordinarily be the upper end of the range of acceptability.” In addition, EPA would attempt to provide protection to the greatest number of people possible at an excess individual lifetime risk of cancer no higher than 1 in 1 million ( $10^{-6}$ ), taking into account additional factors relating to the appropriate level of control (e.g., costs, economic impacts, feasibility). The acceptable risk established in the first step would not be exceeded by the standards EPA adopts based on the second step.

Carcinogens for Which a Margin of Exposure Analysis is Appropriate. Following a determination of nonlinearity, consistent with the guidance in EPA’s proposed revised cancer guidelines (EPA 1996b), an MOE analysis will be undertaken.<sup>1</sup> If such a determination cannot be made, the analysis will default to an assumed linear mode of action and the benzene decision process will be applicable. The MOE analysis will take into consideration the number of people exposed, especially sensitive subpopulations, at the various exposure levels. Individual chemical

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<sup>1</sup> EPA recognizes that the use of an HI approach for non-cancer health effects and a MOE approach for nonlinear carcinogens presents challenges to the economist in performing economics benefits analysis. This concern was also raised in the CRARM report which also discussed a general approach to address the issue (CRARM 1997b, p. 100-101). In the coming years, the scientific community will need to work with economists to devise defensible methodologies for economic analyses of these types of effects.

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assessments of the “appropriate” MOE will be made, considering factors specific to the individual assessment which could include any or all of the following: the steepness of the dose-response curve, persistence of the compound in the body, known human variability in response, and demonstrated human sensitivity as compared with experimental animals. In addition, the chemical-specific MOE evaluation should provide information on the appropriate combination or segregation of the chemicals in the mixture. The use of additivity will be maintained in the absence of data to the contrary. Combination of the chemical-specific MOE values across mixture components will be done by first calculating the ratios of individual HAP exposure levels to the corresponding departure point divided by the chemical-specific “appropriate” MOE.<sup>2</sup> These ratios can be summed for multiple chemicals, and a sum of ratios (i.e., total ratio) greater than 1 may be indicative of a potential hazard. This is roughly analogous to treating the MOE as a UF and calculating a hazard index.

Non-Carcinogens. A verified RfC should be used as the chemical-specific inhalation health criterion in the calculation of the HI. In assessing chronic risks, the HI is based on long-term exposure estimates and is calculated based on target organ effects, where adequate data exist to allow such calculations. For each chemical in the mixture, a thorough review of the toxicity literature may be required to determine which organ systems are affected (e.g., liver, respiratory, central nervous system). For chemicals with no RfC or if the RfC is not verifiable, an appropriate alternate value may be used. While EPA’s non-cancer risk management policy is not yet available, it is expected that a HI less than 1 that is derived using target organ-specific hazard quotients would ordinarily be considered to meet an ample margin of safety. If the HI is greater than 1, then the magnitude of the exceedance of the HI, the uncertainty in the HI, the slope of the dose-response curve, and a consideration of the number of people exposed would be considered in determining whether an ample margin of safety is met.

Evaluation of the allowable extent of exceedance of an HQ or an HI of 1 also would consider the values of UFs and the confidence in the RfCs that are used in the calculation of the HI. In general, it is considered that each UF is somewhat conservative; because all factors are not likely to simultaneously be at their most extreme (highest) value, a combination of several factors can lead to substantial conservatism in the final value. Larger composite UFs lead to more conservative RfCs. Conversely, lower composite UFs are less conservative and lead to a higher level of confidence in the RfC. Intermediate UF values or a mixture of high and low UFs would require an examination of the relative contribution of various chemicals to the HI.

The non-cancer acute HI should be calculated based on effects in the same target system. The acute HI should be calculated based on appropriate short-term exposures. Appropriate UFs for animal to human extrapolation and for data base deficiencies should be used if needed to make the data comparable. The ARE, where available, should be used as the chemical-specific health

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<sup>2</sup> For example, if the departure point for a given chemical is  $5 \mu\text{g}/\text{m}^3$ , the chemical-specific “acceptable” MOE is determined to be 1,000, and the exposure level is  $0.0005 \mu\text{g}/\text{m}^3$ , the ratio for that chemical would  $0.0005 \div (5/1,000) = 0.1$ .

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criterion in the calculation of an acute HI. For chemicals with no ARE, a provisional ARE should be developed. If the ARE is not verifiable, then an appropriate alternate value may be used. These analyses should be done in a manner similar to that discussed above for chronic non-cancer effects. On a case-by-case basis, HIs somewhat greater than 1 may be considered to meet an ample margin of safety based on consideration of the factors described above for chronic HIs.

#### **4.1.2 Available Methods and Costs of Reducing Residual Risks**

The CAA section 112(f)(1)(B) directs EPA to investigate and report on “the technologically and commercially available methods and costs of reducing [residual] risks” from HAPs. The EPA believes that for most source categories there are reasonable options beyond MACT if it is determined that additional control is needed. This section provides an overview of these options, with an emphasis on pollution prevention approaches.

Two general types of strategies can be used to reduce the human health and environmental risk associated with HAP exposure. One is to limit releases into the atmosphere. These “pre-release” strategies employ various control technologies and pollution prevention methods developed by industry to comply with regulations requiring them to reduce HAP emissions. A second approach, applicable primarily to protecting public health, is through the adoption of “post-release” strategies to keep people out of HAP exposure pathways – that is, to eliminate or minimize contact between people and HAP-contaminated media. Measures of this type can include institutional and regulatory approaches such as zoning controls and advisories, which limit public access to areas that contain unhealthful HAP concentrations, fishing restrictions and fish consumption advisories, and provision of alternate drinking water supplies. These strategies are used most often in cases where unregulated sources already have emitted large quantities of pollutants, or as emergency response measures to protect the public from pollution caused by accidents or spills.

Pre-release strategies have traditionally been the preferred method to protect the public from exposure to harmful pollutants because they minimize the impact on the environment, at lower costs over the long term, and place the burden of managing wastes on the source itself. Pre-release methods are consistent with EPA’s environmental management philosophy of encouraging pollution prevention/recycling/treatment first, and pollution disposal/release only as a last resort. Hence, this section focuses on the technologically and commercially available pre-release strategies that can be used to reduce residual risk.

Given the site- and HAP-specific nature of control technology and cost determinations, combined with the fact that there are 188 HAPs and more than 170 source categories and that no post-MACT risk assessments for entire source categories have been completed, an in-depth discussion of the specific methods and costs of controlling post-MACT HAP emissions is beyond the scope of this report. Instead, the remainder of this section presents a brief review of emissions control strategies “beyond MACT” and discusses available options for further reducing the risks of HAP emissions to the general public. A discussion of general MACT requirements is followed by

an overview of currently available control strategies, with an emphasis on ways that industries can go beyond the requirements of MACT and other existing air regulations. Topics addressed include site-specific parameters needed to select appropriate controls for a specific facility and available options for reducing emissions, including add-on control equipment, process/work practice modifications, pollution prevention techniques, and voluntary/incentive based programs that encourage facilities to further reduce HAP emissions. Finally, a general discussion on the key factors that influence the costs of these various strategies is provided.

### **MACT and Beyond**

Emission standards such as MACT may require one or more of the following in order to show compliance: use of a particular control device; meeting a numerical control target; or implementation of a certain work practice or operational restriction. The regulatory limitations will determine what options a source has for demonstrating compliance. After national MACT standards for a particular source category have been promulgated, all affected sources of HAPs must select and adopt controls equal to or more stringent than MACT in order to achieve the necessary emissions reductions. The selection and exact specification of controls is a site-specific determination (as discussed further in the section below entitled “Available Control Strategies”).

The MACT determinations, like other broadly applicable emissions control standards, are based upon decisions about the most effective, feasible, and reliable controls available. It is important to note, however, that MACT standards for existing sources in a particular source category do not necessarily represent the most stringent state-of-the-art controls available to that industry. In fact, new source MACT for a given source type is often more stringent than existing source MACT for the same source type. In some cases, State and local control standards are more stringent than MACT for some source types (e.g., California’s Lowest Achievable Emission Rate (LAER) standards for nonattainment areas).

Cost and other considerations may prohibit the most effective controls from being selected as the national MACT standard. This is required in the statutory language of the CAA, which states that MACT standards

“...shall require the maximum degree of reduction in emissions of the hazardous air pollutants... that the Administrator, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts...determines is achievable...”

Accordingly, controls capable of achieving greater HAP reductions may have been ruled out at the time of MACT determination because of cost or other considerations. However, such costs may later be determined by EPA to be reasonable in the face of significant residual risks. It is also possible that, over time, market conditions or technological breakthroughs in certain control technologies could reduce the cost of currently expensive controls to less prohibitive levels, making their adoption more feasible. Further, it is unlikely that the regulatory standards will be

updated frequently enough to keep up with control technology advances. As a result, there are likely to be new and emerging control options in the future that facilities can use to achieve additional HAP reductions beyond MACT levels of control.

At the time a source category is determined to need additional controls to reduce the remaining risk, specifics of the source stream and the HAPs will be better known. The controls in-place can then be examined both for new and existing sources within the category. One obvious option for further reducing the risk would be to explore application of the new source level of control on existing sources, assuming that less stringent levels of control were required for existing sources in the section 112(d) standards.

### **Available Control Strategies**

The most appropriate HAP control technology for a particular application must be determined on a case-by-case basis after careful consideration of many site-specific issues, such as the design of the facility, the overall manufacturing process, the chemicals being used, the emission stream characteristics, the desired control efficiency, and the cost-effectiveness of the various control options. Even within a particular industry, the tactics used to control a specific type of HAP from a certain industrial process will vary from facility to facility. Because of this considerable variation in the types of controls used, a detailed discussion of specific strategies is beyond the scope of this report. Instead, a review of the general types of control options that are available to facilities to achieve meaningful HAP reductions beyond the MACT requirements is provided.

Different techniques are used to control emissions from each of three major types of emissions sources: process point sources, process fugitive sources, and non-point fugitive sources. Process point sources include industrial processes that discharge emissions through a vent-pipe or stack. Process fugitive emissions include dust, fumes, or gases that leak from pumps, valves, compressors, or other components. Non-point fugitive sources are large surface areas, such as storage tanks and waste treatment pools, from which HAPs are emitted (process and non-point fugitive sources are hereafter referred to simply as “fugitive sources”).

Past efforts to control industrial HAP emissions often have focused on point sources because of the clearly defined nature of the point source emissions stream. By contrast, fugitive emissions, which can include hundreds of individual leaking components at a single facility, are very dispersed, making them more difficult to trap and control. In addition, point source emissions (prior to control) generally represent a much larger percentage of total HAP emissions than fugitive sources. For these reasons, point source control has traditionally been the most cost-effective means to achieve the greatest emission reductions. Unless otherwise noted, the control strategies discussed hereafter apply to point sources.

Traditionally, industrial facilities have complied with air pollution regulations by retrofitting emission stacks with end-of-the-pipe control technologies. As industries have grown, however, so

too has the amount of pollutants generated. While traditional stack controls continue to be a necessary and effective way to reduce emissions, the marginal benefit of investing in stack controls has lessened significantly for many source types, prompting sources to seek more cost-effective control options. In response, source owners have developed a variety of innovative approaches to reduce pollution at the source as a way of avoiding the need for costly emissions control technology. Many of the techniques are designed to achieve emission reductions by improving the overall efficiency of the facility. For example, some facilities have re-designed their production processes to reduce the amount of fuel combusted and waste generated. Others are turning to pollution prevention (P2) tactics, which focus on reducing pollution at the source as a way of avoiding costly add-on controls. Pollution prevention accomplishes this by minimizing waste generation through cleaner production.

Other facilities are starting to use a combination of techniques to more efficiently control emissions where a single control technology may have once been used. Few are using all the methods available to them, however, so even the least-polluting facilities are likely to be able to find additional control strategies with which to supplement their current ones. These tactics are being used with increasing frequency by industry as effective ways to achieve meaningful emission reductions on top of existing controls.

Effective strategies for controlling HAP emissions – many of which will be applicable to further controlling sources already subject to MACT – include:

- Pollution prevention techniques, such as replacing hazardous substances with less harmful substitutes;
- Re-designing production processes;
- Adding a technological control, either to a previously uncontrolled source or as a supplement to existing controls;
- Replacing existing controls with a more effective control technology; and
- Changing worker practices.

Methods range from the complex and costly (e.g., redesigning the manufacturing process or retrofitting stacks with sophisticated technological controls) to less costly P2 approaches (e.g., substituting hazardous substances with less toxic alternatives or modifying work practices to reduce emissions). Facilities can be further encouraged to reduce HAP emissions through the use of voluntary/incentive-based programs. This range of control options is discussed further below.

**Add-on Controls.** Different add-on control technologies are required for point and fugitive emission sources. Fugitive source emissions can be captured with hoods, enclosures, or closed vent systems and then transferred to a control device, such as those noted below. Improved equipment (e.g., pumps, valves, seals) may also be used to prevent fugitive HAP emissions. Different add-on technologies are used to control emissions of organic vapor, inorganic vapor, and particulate HAPs. Add-on devices used to control organic vapor emissions include combustion devices (i.e., thermal incinerators, catalytic incinerators, flares, boilers, and process heaters) and

recovery devices (i.e., condensers and absorbers). The two most common methods available for controlling inorganic vapor emissions are absorption (scrubbing) and adsorption. A third technique, combustion, may be used for some inorganic HAPs (e.g., carbonyl sulfide). The three types of devices typically used to control particulate HAP emissions are fabric filters (baghouses), electrostatic precipitators, and venturi scrubbers. The applicability of each device depends on the physical and/or chemical/electrical properties of the HAP particle under consideration in addition to the specific gas stream characteristics and parameters.

**Process/Work Practice Modifications.** Process modification refers to any strategy that seeks to reduce emissions by changing the operating practices of the facility or making internal equipment changes. Examples include the re-design of a system to recover and recycle the emissions stream. Some firms choose to make internal equipment changes by implementing cleaner processing technologies through equipment modifications and modernization. Many of these strategies overlap with the P2 tactics that are being used with increasing frequency by industry (discussed below). Operating practice changes include re-designing industrial processes to be more efficient, or instituting alternative work practices to reduce emissions. Work practice changes may include a wide variety of activities such as changing the ways that employees apply industrial solvents or reducing the amount of solvents used and allowed to evaporate. Also, where workers are directly involved in a manufacturing process there may be ways to change worker practices to reduce HAP emissions. Another example is increasing maintenance of process equipment. Implementing a leak monitoring program to detect and repair leaking components is an effective worker practice to reduce fugitive emissions.

**Pollution Prevention.** Pollution prevention is the catch-phrase used to describe a set of control strategies designed to minimize waste generation through cleaner production. The Pollution Prevention Act of 1990 defines P2 as any source reduction practice that “reduces the amount of any hazardous substance, pollutant or contaminant entering any waste stream or otherwise released into the environment (including fugitive emissions) prior to recycling, treatment, or disposal.” The potential benefits of P2 strategies include improving plant efficiency, saving money, and enhancing the quality and quantity of natural resources for production. In addition, P2 can be more cost-effective than traditional add-on HAP controls. While there is much discussion and debate about what exactly constitutes P2, the following general characteristics are typical:

- Reduction of substance volumes;
- Substitution for toxic substances;
- Implementation of clean technology; and
- Installation of in-process recovery equipment (recycling).

Reducing the amount of toxic chemicals used in the production process generally results in cleaner production and the generation of less waste, including HAPs. Product substitution involves replacing hazardous substances used in the production process with alternatives that result

in lower HAP emissions. A common example is the replacement of VOC-laden solvents and lubricants with water-based formulations. Many emission-producing chemicals used in manufacturing have environmentally safe substitutes that can be used in their place. In some cases there may be effectiveness and cost trade-offs to using an alternative product, but for many industrial substances cost-effective alternatives exist. Ultimately, each of these P2 programs reduces the amount of wastes that are generated in the production process. Because the combustion of industrial wastes is a major source of HAP emissions, designing facilities to produce less waste will result in direct air quality benefits.

**Voluntary and Incentive-based Approaches.** More industries than ever before are voluntarily controlling emissions. This is due in part to the many Federal pollution prevention programs that have been established to encourage self-regulation by industry, as well as to liability considerations, community pressures, and the desire for a favorable public image. For several years EPA has been experimenting with voluntary partnerships between government and industry as a means to more rapidly achieve environmental goals. The EPA's 33/50, Energy Star, Green Lights, and Green Chemistry programs have succeeded in gaining commitments from thousands of industrial sources to reduce air emissions, including HAPs. Industries have responded positively to these programs because of their voluntary nature and the positive public recognition they receive for participation. Some have even reported other benefits, including quicker turn-around on their permit applications. Their success in achieving environmental results demonstrates that voluntary programs can be an effective way to encourage companies to adopt control strategies for reducing HAP emissions and residual risks.

Incentive-based policies may be another way to reduce the total HAP emissions released into the atmosphere beyond currently mandated MACT levels. These policies allow sources the flexibility not only to choose what technologies to use for their reductions, but how extensive their reductions will be. Decisions about the extent of reductions are made in response to market-like signals: achieving large emission reductions can bring monetary rewards, while foregoing emission reductions is permissible, though at a price. A well-known type of incentive program is the cap-and-trade program, in which all affected sources are provided with a strictly limited (or "capped") number of transferable emission allowances. The sources are allowed to emit one unit (e.g., a ton or a tenth of a ton) of the regulated pollutant for each allowance they hold. Any source that finds it preferable to emit more units than they were provided allowances for must purchase additional allowances from another source. Because the source that sells some of its allowances must reduce its emissions below the amount originally allocated to it, total emissions are kept within the cap.

### **Control Strategy Cost**

Just as specific control technologies cannot be examined until the source category and HAP have been identified, the specific cost to reduce any residual risk that may remain following MACT implementation cannot be determined at this time. Cost analyses are critically dependent on numerous and various conditions, including individual source stream characteristics, HAP characteristics, site conditions at a particular facility, level of control necessary, and the various

control options that may be considered. After MACT has been promulgated and a source category and particular HAP (or HAPs) have been identified for residual risk reduction, a detailed cost analysis can be performed.

Factors that may be considered in assessing the cost-effectiveness of a particular control strategy include:

- Capital costs (i.e., the cost of the equipment, estimated costs for site preparation and installation, and cost of ancillary modifications and upgrades to monitoring and process control equipment);
- Cost of capital for the affected industry;
- Fuel costs;
- Chemical costs;
- Incremental labor costs to operate equipment;
- Production penalties associated with the equipment, and other opportunity costs;
- Control efficiency for various streams;
- Expected performance degradation over the life of the equipment;
- Adjustments for tax treatment of equipment; and
- Expected equipment life.

With this information, capital costs can be annualized; operating costs can be disaggregated into fixed and variable costs; life cycle, annual emission estimates can be derived; and costs and emission reductions can be estimated for a variety of operating scenarios. These data are typically put into an existing model, such as the EPA model HAP-PRO, in order to determine control cost effectiveness in terms of cost per mass of pollutant reduced.

## **4.2 Section 112 (f)(1)(C)**

### **4.2.1 Actual Health Effects and Epidemiological Studies**

Section 112(f)(1)(C) requires EPA to assess and report on "the actual health effects with respect to persons living in the vicinity of sources, any available epidemiological or other health studies..." Information on actual health effects on neighboring populations resulting from HAP emissions from source categories is limited. This section presents a summary discussion of epidemiological, laboratory, and other exposure studies, then briefly describes how the EPA intends to use these data and actual source category-specific health effects data that may become available in the context of section 112(f) residual risk assessments.

#### **Current State of Knowledge**

The earliest efforts to investigate the relationship between air pollution and ill health were focused on characterizing the relationship between obvious and acute effects (respiratory irritation, exacerbation of asthma, other respiratory and cardiovascular disease and death) and short-duration

incidents ("air pollution episodes") of high exposures to combustion products. Beginning in the late 1980s, studies of adverse health effects near hazardous waste disposal sites began to appear, including U.S. studies such as those conducted by the Agency for Toxic Substances and Disease Registry (ATSDR), as well as a number of foreign studies. While it has been reported that individuals who live or work in the vicinity of sources of air toxics emissions were, in some cases, found to have higher exposures than the general population, most health effects studies, generally, do not focus on populations near sources of HAPs. Therefore, information on potential health effects of air toxics is primarily based on laboratory animal and occupational studies. These types of studies are suggestive of potential adverse effects, but usually evaluate chemicals at higher exposures than normally expected for human populations. Human data give evidence of potential effects, but are often limited by lack of actual exposure conditions, lack of statistical power, or confounding factors.

Besides laboratory and occupational studies to assess health effects, investigators have employed techniques such as follow-up studies of geographic patterns of disease (particularly cancer), emissions inventories, exposure and risk assessment studies, and biomarker studies of selected pollutants (see accompanying text box). These studies generally have focused on the following major types of health effects – cancer, respiratory irritation and other respiratory toxicity, neurobehavioral toxicity, hepatic effects, renal effects, and reproductive and developmental effects – attributed to air pollutants, and investigators have evaluated associations between exposures and health effects. For example, epidemiologic studies of air toxics have focused on the cancer endpoint because (1) there are established and easily accessible databases of cancer mortality and, to a lesser extent, incidence at national and regional levels, and (2) many toxic air pollutants are suspect or confirmed human carcinogens. Some of these carcinogenic pollutants also are convenient subjects for environmental studies because they are persistent in air and soil-water systems, and exposures can thus be more readily measured and estimated.

Focused studies of particular classes of toxic air pollutant sources to assess effects of adverse exposures have also been performed. Initially, attention was given to the well-studied and common metallic pollutants such as cadmium and lead and the other criteria pollutants, or other general indicators of air quality. Some of the toxic metals represent special cases, each having its

**SOME APPROACHES TO ESTABLISH RELATIONSHIP  
BETWEEN AIR TOXICS EXPOSURE AND HEALTH EFFECTS**

**Laboratory studies.** Adverse health effects of exposures to specific pollutants are often evaluated in studies with laboratory animals or human volunteers. In these studies, the pollutant concentrations are likely to be higher than the exposures to the general population.

**Studies of geographic patterns of disease incidence or mortality.** Studies of vital statistics, disease incidence, or mortality may disclose geographic patterns of adverse health effects that are suggestive of a relationship to specific pollutants or pollutant sources. If such studies are not supplemented by exposure data, and are not controlled for confounding factors other than pollutant exposures, it is not possible to support inferences of causation associated with pollutant exposures.

**Studies of general population exposures, exposure indices, and biomarkers.** These types of studies have been used to estimate human exposures to pollutants and draw inferences about potential adverse effects. The collected information is often used, in conjunction with toxicity data, to conduct risk assessments. In some instances, measurable indices of exposures (biomarkers of exposures), such as body burdens or tissue concentrations of pollutants, can be used to document exposures and evaluate the potential for adverse effects.

**Occupational exposure/epidemiology studies.** Health effects of specific pollutants are often first discovered through observations of adverse effects in workers exposed to high levels of the pollutants. These studies, however, do not directly address the potential for adverse effects occurring in the general population at lower exposure levels.

**Formal environmental epidemiology investigations.** A "formal" environmental epidemiology study involves systematic investigation of the relationship between an observed pattern of adverse health effects and exposures to one or more agents. The analysis of actual (as opposed to estimated) health outcome information is what distinguishes an epidemiological study from a risk assessment or a biomarkers study. Systematic efforts to control for confounding factors (factors other than exposures to the toxic substances of interest which may be responsible for the observed effects) are what distinguish a formal ("analytical") epidemiologic study from a simple "descriptive" summary of geographic patterns of disease incidence. Often, formal epidemiologic studies are not a powerful enough tool to discern relatively small increases in disease.

**Risk assessments.** In a risk assessment, information about exposures (which may reflect actual measured exposures or exposures estimated using emissions and environmental models) is combined with toxicity information (from occupational or laboratory studies) to develop predictive estimates of the frequency or severity of occurrence of adverse effects in human populations. There is a high degree of uncertainty due to imprecision in exposure estimates and uncertainties in dose-response information, especially at low doses.

own unique pattern of non-cancer effects. The renal effects of cadmium exposures, (ATSDR 1993a), neurodevelopmental impacts of lead (ATSDR 1993b), and reproductive toxicity of mercury exposures (ATSDR 1994) are the most well-studied examples. In addition, a few studies use total mortality, or cause-specific mortality, as endpoints. Individually, these various studies have provided data that contribute to an understanding of the relationship between air pollution exposure and adverse effects, on both the qualitative and quantitative level.

The EPA has recently surveyed the published literature on the actual human health effects of outdoor air toxics exposures at ambient levels (EPA 1995e), and some information from this study is summarized in this section and provides examples of the difficulties inherent in making causal connections between exposure and effects. One of the most extensively investigated connections between exposure to air pollutants and health effects is that between lung cancer and exposure of populations near smelters to arsenic. Several studies have addressed this relationship

(Brown et al. 1984, Frost et al. 1987, Pershagen 1985). These studies tend to show increased risk associated with exposure (or exposure surrogates, such as distance from the smelter), although the apparent increase was not statistically significant in all cases. For example, Frost et al. (1987) found that lung cancer patients were more likely to live close to an arsenic-emitting smelter (borderline statistical significance) in a case-control study that was conducted with women only in order to reduce confounding from occupational exposure. However, there was no control for smoking and no effect was seen in the cross-sectional phase of their study. Pershagen (1985) analyzed lung cancer data near an arsenic-emitting smelter, with the data stratified by smoking status and occupational exposure. In the group that was not occupationally exposed, there was an increased relative risk with proximity to the smelter for both nonsmokers and smokers, but the increase reached statistical significance only among the smokers. Hughes et al. (1988) reviewed more than 10 studies investigating health effects (primarily lung cancer) in communities near arsenic-emitting industries. They noted that about half of the studies reported significant increases in adverse effects while about half of them reported no effect or decreased risk in the exposed populations. However, these authors noted that many of the studies (particularly those that observed no statistically significant effect) lacked sufficient statistical power to detect the small increases in risk that would be expected, and suggested that some small increase in risk is likely.

With respect to other effects, Nordstrom et al. (1978) found decreased birth weight in babies born to mothers who lived close to an arsenic-emitting smelter. However, it is unclear if the magnitude of the decrease was clinically significant (Hughes et al. 1988).

Several studies have attempted to show an association between vinyl chloride emissions and central nervous system birth defects (Edmonds et al. 1978; Rosenman et al. 1989; Theriault et al. 1983). While all of these studies reported some association between potential exposure and disease, each was limited by uncertainties in the exposure estimates, implausible results, or potential confounding factors such as smoking or drinking. Overall, these studies provide insufficient data to conclude that there is a causal relationship between ambient air exposure to vinyl chloride and central nervous system birth defects.

An overall view of the epidemiologic literature on exposure to air toxics in the environment is consistent with the notion that concern is warranted. However, understanding of the risks to individuals living near sources and exposed daily to these air toxics is limited or confounded by other factors. Except for a few well-known cases (the sudden release of a large volume of methyl isocyanate in Bhopal, India, for example) where extremely high exposures to accidental releases of industrial chemicals resulted in severe acute health effects, the adverse effects of exposures to airborne hazardous chemicals are generally very difficult to detect.

Because of the difficulties in the extent and usability of epidemiology data, the EPA has looked into other types of data that may help bridge the gap between cause and effect. In this context, the state of the art in exposure monitoring and the use of biomarkers has become an expanding field of research. For example, the existing literature on neurobehavioral effects of toxic air pollutants is dominated by discussions of the adverse effects of lead on intellectual and

behavioral indices in children. These studies generally describe decrements in performance as a function of biomarkers of lead exposure, such as blood lead concentrations or heme metabolite levels. There is, however, little information available from these studies on the sources of lead exposures, and lead from deteriorating paint and in pipes and solders used for drinking water distribution can contribute significantly to total exposures.

In a study by Binkova et al. (1995), PAH DNA adducts were measured in a group of women in the Czech Republic who worked outdoors for about 8 hours per day. Personal exposure monitoring was used, allowing both indoor and outdoor exposure to PAHs to be evaluated; exposure to respirable particles ( $<2.5\ \mu\text{m}$ ) and PAHs was measured. Levels of DNA adducts in white blood cells were increased immediately after days of high PAH exposure. This study demonstrated that DNA adducts can be used as biomarkers of exposure, reflecting short-term exposure levels. In addition, DNA adducts can be used as biomarkers of effect, because, if unrepaired, they can lead to gene mutations, which in some cases can ultimately lead to cancer. However, due to the multiple steps from gene mutation to cancerous cell, DNA adducts and gene mutations are best viewed as indicating carcinogenic potential rather than indicating actual risk of cancer.

Blood or tissue concentrations of metals such as cadmium are also occasionally used as indicators of exposure and potential adverse effects for airborne toxics. Among the studies that use biomarkers of exposure are evaluations of tissue, hair, and urine cadmium levels in a population near heavily industrialized cities in Russia (Busteva et al. 1994). Urinary cadmium is a reliable indicator of recent cadmium exposure, as shown by several occupational studies. The presence of the protein  $\beta$ -2-microglobulin in urine (termed proteinuria) is also considered a reliable indicator of cadmium exposure. Busteva et al. (1994) reported that the percentage of factory workers having elevated levels of this protein in their urine ( $>250\ \mu\text{g/l}$ ) was highly correlated with the air content of cadmium. Although no significant effect was seen in the general population, this may have been due to the small sample size and resulting low statistical power. Proteinuria ( $\beta$ -2-microglobulin levels above  $250\ \mu\text{g/L}$ ) was observed only in the exposed worker population. Collecting biological samples and conducting laboratory testing, as in this study, is more labor-intensive than doing epidemiological investigations using disease registries. However, because proteinuria is a well-characterized effect of cadmium exposure, and both exposure and effect biomarkers can be monitored by urinalysis, this technique has applicability where high exposure to cadmium is expected.

Another potential source of information may be nationally standardized and comprehensive disease registries or databases for adverse effects of toxics exposures, such as birth defects and reproductive outcomes (Shy 1993), but again, there are limitations in its use. Currently, studies that use these sources require investigators to obtain access to local or State health status information, whose availability is highly variable from State to State, or to obtain information from hospital or other medical records where confidentiality may become an issue. This difficulty is less of a concern for case-control studies, but can severely limit the ability to do large-population cohort analyses or cross-sectional studies.

Acute effects such as seen in occupational settings are less likely to be seen in the general population exposed to toxic air pollutants at ambient levels, with the possible exception of chemicals that have specific irritant properties. In addition, the effects of usually low chronic exposures to toxic air pollutants may be subtle, and may develop slowly over time in response to cumulative exposures (chronic effects), or may not develop until long after exposures occur (latent effects). Information on exposure levels to toxic air pollutants near sources, as well as to "background" pollutants that may be confounding the results of air pollutant epidemiology studies, is also generally limited. Thus, it is not easy to directly characterize the risks associated with general population exposures to toxic air pollutants under conditions of chronic low-level exposures. Nonetheless, it is currently assumed for prudent public health reasons that such effects may be occurring because, for example, many toxic air pollutants are suspected or known human carcinogens and even low levels of exposure could theoretically cause increased cancer risks. In a smaller number of cases, animal or controlled human studies indicate that noncarcinogenic effects might be expected to occur at exposures near ambient levels. In some instances, allergic sensitization may result in adverse effects in a small, especially sensitive subset of the exposed population. There is presently no national monitoring system for air toxics that can provide even general information on the urban and rural concentration patterns of these pollutants in ambient air.

Other issues to consider in trying to assess the actual health effects of air toxics include (1) the lack of indoor exposure data and (2) the often-observed coincidence between exposures to toxic air pollutants and exposures to criteria air pollutants. Information on indoor exposure data is useful since the majority of individuals spend most of their time (usually 80 percent or more) indoors. Because pollutant concentrations in indoor air tend to be quite different from (and often higher than) those outdoors, studies which do not take indoor air quality into account will have difficulty in elucidating the true relationship between air toxics exposures and effects. Both toxic air pollutants and criteria pollutants are associated with areas of high population density and industrial development, and many epidemiologic studies simply use measures of one or a few of the criteria pollutants as the sole measure of exposure, and use it as a proxy for all "air pollution." For example, in many studies that assess the relationship between particulate exposures and acute and chronic health effects (usually where there is no clearly identified dominant source of particulate air pollutants), it is not known which chemical constituents of particulates contribute to the observed increases in risk, and it is therefore difficult to attribute any given fraction of these effects to toxic air pollutants.

### **Strategy for Considering Actual Health Effects/Epidemiology Information in Residual Risk Analyses**

Early in the data gathering stage, the EPA will search the scientific literature for published epidemiological studies related to the specific source categories, HAPs, and/or locations studied. These reports will be evaluated for quality, with preference given to those covering emissions from the source categories of concern at environmentally relevant concentrations over long periods. Where published epidemiological studies are unavailable, the EPA may also consider, as part of its

refined analysis, examining other types of available human health data for possible correlations between exposure and adverse effects. Potential sources of health effects information include State or national disease registries (e.g., the Centers for Disease Control's Birth Defects Monitoring data base), hospital and other medical records, death certificates, and questionnaires. The EPA intends to coordinate the identification, collection, and review of such data with the Public Health Service and other Federal, State, and local public health officials. Examples of widely reported outcomes include cancer incidence or mortality, birth defects, and respiratory symptoms. Information on pollutant-specific biomarkers – biological measurements associated with exposure to certain pollutants – may also be available. Exposure to HAPs may be estimated in several ways, including ambient monitors, mathematical modeling, or personal air monitors. The EPA recognizes the difficulties that exist in obtaining actual health effects data. However, the EPA believes that it may be useful to incorporate some kind of health effects/epidemiology data in the residual risk assessments for selected air pollutants and source categories and intends to use existing data wherever scientifically appropriate. Clearly, any actual health effects data can generally only be used to help establish current or past conditions, and cannot be used directly in the prediction of post-MACT risks that may occur in the future (i.e., residual risks).

#### **4.2.2 Background Concentrations**

Section 112(f)(1)(C) also requires EPA to assess and report on "risks presented by background concentrations of hazardous air pollutants..." This section of the report discusses general information on background levels, including the cumulative risk policy in development, and presents a definition of background concentrations for air toxics and residual risk purposes. It describes approaches used by other EPA programs and includes examples of rules and guidance that consider the issue of background. It also presents a discussion of the difficulties in addressing background concentrations in residual risk analyses and identifies data needs to assess background. The section concludes by describing EPA's options to analyze and consider background concentrations in residual risk analyses. It describes how EPA will assess available monitoring data for individual source categories under study, and how background concentrations will be evaluated in residual risk assessments and treated in decision-making.

Background concentrations may be considered to be the levels of contaminants that would be present in the absence of contaminant releases from the source(s) under evaluation. Background concentrations come from contaminants that either may occur naturally in the environment or originate from anthropogenic sources. Background contamination can be localized or ubiquitous. An example of localized contamination is the presence of high concentrations of trace metals in dust from geologic formations naturally high in trace metals. An example of ubiquitous contamination is the widespread presence of low concentrations of polycyclic aromatic hydrocarbons in soil and dust in areas near forest fires.

The EPA's Science Policy Council is developing a cumulative risk policy with the goal of developing a framework for conducting cumulative risk assessments. While Part 1 of the Guidance on Cumulative Risk Assessment released in August 1997 (EPA 1997e) does not provide an explicit

definition of cumulative risk or background, in general cumulative risk is considered to include risks from multiple sources, pathways, and pollutants. The cumulative risk guidance identifies elements that must be considered in a cumulative risk assessment such as the cumulative effects of mixtures on different and the same target organs from multiple sources by direct and multipathway exposures. Cumulative risk is therefore broader than the "incremental risk" (or "excess risk") attributable to a given source/pathway/pollutant combination under evaluation.

Presently, EPA does not have comprehensive Agency-wide guidance or policies on incorporating background concentrations into air toxics risk assessments and risk management decisions. EPA's general approach in previous risk assessments and risk management decisions has been to assess incremental risk of a particular source or activity and compare that risk to an "acceptable risk" criterion (or set of criteria). Thus, there is no explicit Agency-wide guidance that can be referenced for a working definition of background concentrations or risks for the residual risk program. However, based on previous actions within various EPA programs, some direction can be inferred. For example, approaches to considering background risks can be inferred by the precedents set by various documents and risk assessments written by other EPA offices. Summarized below are several major EPA programs and rules where background concentrations and risks are considered.

#### **EPA Programs and Rules that Consider Background Concentrations and Risks**

Site risk assessments under Superfund and the RCRA corrective action program require the collection of background samples at or near hazardous waste sites in areas not influenced by site contamination, but that have the same basic characteristics as the medium of concern. Generally, comparison of background and source-related contamination is used to identify areas affected by the source and contaminants attributable to the source. Incremental risks are then assessed for contaminants in media demonstrated by comparison with background concentrations to have originated from the source. The level of risk reduction is generally set by cleanup levels based on achieving an acceptable risk or reducing contaminants to background concentrations, whichever is least stringent. However, in some cases where anthropogenic background levels exceed cleanup goals, EPA may determine that a response action under Superfund is necessary and feasible, and a comprehensive plan may be developed to address area-wide contaminated media not originating from the site source. In such cases, reduction of anthropogenic background risks becomes an additional goal of the remediation program.

In 1993, the Office of Wastewater Management developed a comprehensive risk-based rule, known as the "Part 503" rule, to protect public health and the environment from the anticipated adverse effects of pollutants that may be present in sewage sludge that is applied to land. Using the results of the rule's multipathway risk assessment that considered soil background metal concentrations in the calculations of risk-based pollutant concentration limits, EPA set pollutant concentration limits above which sludge could not be applied. The limits were derived by calculating the increment of pollutant from sewage sludge that could be added to the total background receptor intake or plant uptake without exceeding a threshold dose. For human

receptors, the threshold dose was set for non-carcinogens at the chronic effects reference dose, and for carcinogens, at an incremental individual lifetime cancer risk of  $10^{-4}$ . For non-human and plant receptors, background soil concentrations were subtracted from reference adverse effect concentrations to calculate the increment of a pollutant from sewage sludge that could be applied to soil without adverse impact. In short, soil-related background concentrations and risks were directly and quantitatively considered in this risk management decision.

The Office of Water developed methods to set maximum contaminant level goals (MCLG) at concentrations at which no known or anticipated adverse health effects occur. Drinking water equivalent levels (DWEL) are calculated from reference doses by assuming a specific receptor body weight and consumption rate. The MCLG is set by multiplying the DWEL by the percentage of the total daily exposure expected to be contributed by drinking water (i.e., the "non-background" portion), called the relative source contribution (RSC). Generally, EPA assumes that the RSC from drinking water is 20 percent of the total exposure, unless specific exposure data for a chemical is available, and that 80 percent of exposure comes from other sources. The RSC may be as high as 80 percent. The EPA also is using this approach of reserving a portion of risk to background in setting pollutant limits covered by the Food Quality Protection Act (FQPA) and in the Office of Pesticide Program's re-registration decisions.

The EPA has not addressed in detail the issue of background risks or cumulative risks in its RCRA hazardous waste listing determinations. In a recent hazardous waste listing determination for petroleum refining process wastes, EPA conducted analyses that considered multiple wastes disposed in land units (wastes with similar constituents from other sources) and multiple units at a facility, thus accounting for the impact of certain other background sources.

### **Difficulties in Addressing Background Risk**

The EPA's lack of a generalized approach to considering background risk in its risk assessments and risk management decisions is demonstrated by the absence of discussion of background risks in many of its major rules and the simplified approaches used in rules that consider background concentrations. One major difficulty comes from the fact that accounting for all possible sources and routes of exposure to pollutants with similar toxic mechanisms is a complex and expensive task with many variables requiring much input data. Methods used to assess risk are evolving and new, more sophisticated models and strategies to assess multiple pathways of exposure are being developed. These models require many variables to accurately account for all sources of background risk, at least some of which are not likely to be available. Lack of data and funds required to collect the extensive data needed to assess multiple direct and indirect pathways has often resulted in the use of simplified assumptions and models such as limiting assessments to direct exposure pathways and regulatory decisions that set background contributions to conservative default values. What is considered background risk is also affected by the approach taken to define a source (e.g., whether the assessment of risk is performed on a source category basis or a point source basis).

Data from the EPA's cumulative exposure project, which is developing a national distribution of estimated cumulative exposures to HAPs, may help to partially fill some of these data gaps. However, background concentrations are not static. The half-lives of contaminants are wide ranging and must be considered when assessing risks over a period of time. Persistent and bioaccumulating contaminants moving along the foodchain alter background concentrations over time. The exchange of contaminants between media (e.g., particulate deposition in surface water) also introduces a time-related background change. In addition, regulatory changes that reduce releases of contaminants from sources will alter background concentrations over time. For example if drinking water standards (or other standards affecting exposure) are lowered, cumulative background risks are also lowered. Therefore, if the approach to managing residual risks of HAPs ultimately accounts for a portion of the risk from background, then a lowering of the drinking water contribution (or other contributions) to background risk potentially could allow for less reduction in residual risk from HAPs, while achieving a similar level of overall protectiveness. However, given the considerable uncertainties in risk assessment generally, it is not clear that a thorough consideration of background, even if possible, would greatly improve the overall conclusions of the assessment.

### **Defining Background for Residual Risk Analyses**

Given the complexities associated with assessing cumulative risk from all chemicals and sources, background concentrations and risks for residual risk analyses will be assessed whenever possible on a chemical-by-chemical basis for the particular HAPs under evaluation. Although other chemicals may contribute to the cumulative background risk because of interactions or effects on the same target organ, the data needed to evaluate cumulative risks from multiple chemicals would be quite extensive and difficult to collect. Thus, background concentration of a particular HAP for either an affected source or source category under evaluation is defined as the concentration of that particular HAP in environmental media attributable to natural and anthropogenic sources – both on-site and off-site – other than the source being evaluated. As described above, background concentrations may change over time, and analysis of background risks would be more accurate if these changes in background concentrations were accounted for. However, because of analytical complexity (e.g., data needs, modeling difficulty, high uncertainty), background concentrations generally will be based on a given point in time when taken into account for residual risk analyses.

Therefore, for the residual risk program, background concentrations will be considered from two perspectives: the contribution of HAPs from natural sources, and the contribution of HAPs from all anthropogenic sources other than the source under evaluation. For a particular point source at a facility, for example, the contaminants present in air in the absence of the source under evaluation may originate from natural sources as well as from other on-site and off-site emissions sources. It follows that the background risk is the cumulative risk from all possible natural and anthropogenic sources of a HAP other than the particular source or source category under evaluation. Residual risk will be assessed in the context of both kinds of background when the sources can be identified and their contributions measured and compared.

### **Strategy for Considering Background in Residual Risk Analyses**

Residual risk analyses will assess incremental risk above background risk, and then assess the significance of these risk estimates using acceptable risk criteria developed and used historically by EPA for judging incremental risk. As described in this report, residual risk will be addressed in a two-tiered approach. In the relatively simple first-tier (screening-level) analysis, the residual risk analysis generally is performed without considering background at all. At most, local or regional scale estimates of background concentrations based on statistical analyses of monitoring data or screening-level modeling analyses (such as air concentration estimates developed in EPA's cumulative exposure project) may be considered. This screening analysis is conducted using conservative methods and assumptions and results are compared to acceptable risk criteria. Where first-tier residual risk estimates exceed the criteria, a more detailed second-tier risk analysis is conducted. In general, an in-depth modeling analysis of background concentrations will be beyond the scope of the refined analysis, although any available background concentration data or other relevant information would be considered. As discussed above, a detailed analysis of background concentrations typically would require extensive data gathering and modeling beyond that required for the incremental risk analysis. For example, numerous nearby (and possibly distant) HAP sources of varying types would need to be characterized in sufficient detail to support release and exposure modeling. In some cases, background risks from HAPs potentially could be considered to play a critical role in evaluation of the need for further reduction of the incremental risk. Thus, EPA may determine that for some source categories, or some individual sources, detailed analysis of background concentrations is warranted.

In such cases, the relative contribution of background to the total risk from HAPs would be considered in decisions for more stringent regulation and may influence the level of reductions required to obtain an "ample margin of safety." If the relative contribution of background risk is high compared to the incremental residual risk, additional source risk reduction may provide relatively negligible benefit. Alternatively, a high relative contribution to total risk by the incremental risk might strengthen the rationale for requiring more stringent regulation. As described above, EPA has reserved part of the "risk burden" for background risk in other regulatory programs (e.g., drinking water and pesticide programs), and this kind of approach will be considered in residual risk decision-making for HAPs.

The data needs for assessment of background concentrations may differ depending on whether a source category or a specific source is under evaluation. For a specific source, identifying the background concentrations from other natural and anthropogenic emissions sources within a specified radius of the source will usually be considered sufficient to demonstrate the relative contribution of background to overall risk and the impact of the single source relative to other sources surrounding it.

### 4.2.3 Uncertainties

This section responds to the CAA section 112(f)(1) requirement to address “any uncertainties in risk assessment methodology or other health assessment technique,” with a focus on uncertainty in residual risk assessments. Uncertainty exists in all areas of risk assessment. Uncertainty in the dose/response estimate can lead to an overestimation or underestimation of the potential risk to the exposed population. For example, the assumption of low dose linearity may tend to overestimate the dose/response estimate if the true shape of the relationship is curvilinear. Another area of uncertainty is that EPA extrapolates cancer risks from occupationally exposed populations (generally healthy white males) to the general population in which susceptibility to a carcinogenic effect could differ. Such susceptibility can differ with age, gender, race, genetic variability, and general state of health. Thus, the dose/response estimate can underestimate the cancer risk to more susceptible subgroups (EPA 1988a, p. 28506). The major inputs to exposure estimation also are subject to uncertainty. For example, emission and plant parameters often must be estimated rather than measured, particularly in determining the magnitude of fugitive emissions. This can lead to overestimations or underestimations of exposure. Similarly, meteorological data are not available at specific plant sites, but are usually available from the closest recording weather station that may or may not be representative of the meteorology of the plant vicinity. In addition, for sources located in complex terrain where the surrounding topography is at higher elevations than the emitting stack, the usual assumption of flat terrain would tend to underestimate the maximum annual predicted concentration of the HAP (EPA 1988a, p. 28507).

Implicit in EPA’s obligation to evaluate the residual risks from releases of HAPs under section 112(f) is the obligation to evaluate the level of confidence that can be placed in the estimates, and the extent to which the numerical estimates of risks might be inaccurate, biased, or unreliable. The systematic consideration of these issues is often described using the general term “uncertainty analysis,” but many different methods are subsumed under this heading, so this term conveys little specific information. The term also can be misleading, because *uncertainty* has a specific meaning in the context of risk assessment, namely “a lack of knowledge about specific factors, parameters, or models” (EPA 1997a). Uncertainty arises from ignorance of fundamental processes, or from lack of data, and uncertainties in risk estimates can be reduced by gathering additional information or by additional scientific research.

The other important part of the problem is the need to address *variability* of key parameters and models and how they affect the risk estimates. In contrast to uncertainty, variability has nothing to do with data quality or knowledge of fundamental relationships, but instead “refers to observed differences attributable to true heterogeneity” (EPA 1997a) in the variables. Examples might include variations in hourly wind velocity, or in the body weights among an exposed population. Beyond a certain point, variability cannot be reduced by data gathering or refinements in models (although it can be more fully characterized). In its 1994 report, *Science and Judgment in Risk Assessment*, the NRC recommended that, when possible, uncertainty and variability should be quantified and the distinction between them maintained throughout risk assessment (NRC 1994).

Other taxonomies have been developed to identify the factors that contribute potential variations in risk estimates as a function of specific types of inputs and models, and the various methods for evaluating these contributions. Some of these specific approaches are discussed in more detail below, but the fundamental distinction between uncertainty and variability analyses is the most important one. For purposes of brevity, we use the term uncertainty in the specific sense to refer to the lack of knowledge about specific parameters and models, and in the more generic sense to describe the overall lack of certainty in risk estimates, which includes contributions both from variability and uncertainty.

In the context of residual risks, uncertainty analysis has important implications both for risk assessment methods and for risk management. Reliable estimates of residual risks cannot be developed unless both uncertainty and variability are adequately addressed, and defensible risk management decisions cannot be made unless risk managers are presented with appropriate information related to the uncertainties in risk estimates. Among the key methodological issues that arise in the context of residual risk are:

- Have all the important sources of uncertainty and variability in the models and input variables to the risk assessment been identified?
- What are the appropriate methods to evaluate uncertainty and variability, given the needs of the decision-making process, the capabilities of available models, and the data and resources that are available?
- What additional data or model refinements can be used to reduce uncertainty in the risk estimates?
- How can information about uncertainty be summarized and presented to decision-makers?

From the risk management perspective, important issues associated with uncertainty analysis may include:

- What is (are) the most useful indicator(s) of uncertainty in risk estimates (from a risk management standpoint)?
- What is the overall reliability of a specific risk estimate?
- What is a reasonable range over which the risk estimate might vary?
- What is the level of certainty that the residual risk estimate is actually greater than zero or less than a defined level of concern?
- How reliably can the relative risks be compared? How well can risks be ranked?

Clearly, risk assessment and risk management issues overlap. In the discussions that follow, the importance of adequate communication between the risk assessors and EPA risk managers is stressed. The development of appropriate methods for uncertainty analysis and defensible decision-making are inextricably linked.

### **Approaches to Addressing Uncertainty and Variability in the Estimation of Residual Risks**

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The EPA has long recognized the need to consider uncertainty and variability in risk assessment. Agency guidance on these issues has gradually evolved over more than a decade, with major documents including:

- Initial set of risk assessment guidance documents (e.g., EPA 1986a,c);
- Risk Assessment Council (RAC) guidance (“the Habicht Memorandum,” EPA 1992d);
- Guidelines for Exposure Assessment (EPA 1992c);
- Policy on Risk Characterization (“the Browner Memorandum,” EPA 1995a); and
- Policy for Use of Probabilistic Analysis in Risk Assessment (EPA 1997b) and Guiding Principles for Monte Carlo Analysis (EPA 1997a)

The Agency has also recently released a revised version of the Exposure Factors Handbook that supports probabilistic approaches to the treatment of a number of commonly employed risk assessment input variables (EPA 1997d). Among these documents, the 1992 Exposure Assessment Guidance, 1997 Policy for Use of Probabilistic Analysis, and 1997 Guiding Principles for Monte Carlo Analysis provide the most detailed recommendations for uncertainty and variability analysis. The former document primarily provides technical guidance on uncertainty evaluation in the context of exposure assessment, while the latter two provide refined technical guidance, as well as recommendations on presentation of uncertainty information to decision-makers. The 1997 Policy also documents EPA’s judgment that probabilistic methods should be used wherever the circumstances justify these approaches. Thus, EPA is committed to carefully considering use of quantitative methods for evaluating uncertainty and variability in its residual risk assessments.

While the exact approach to be taken has not been finalized, several general approaches may be considered for addressing uncertainty and variability in residual risk assessments. There are several basic approaches to uncertainty and variability assessment that are available and may be employed, depending on the nature of the risks being assessed.

**Qualitative Assessment.** Under this approach, the assessor calculates point estimates of risks, and provides only a verbal description of the possible effects of uncertainty and variability on risk estimates. This was the common default approach employed before quantitative methods and the data to support them became more widely available. (Often, a major element of the qualitative discussions of uncertainty is a disclaimer about how little is known about the overall uncertainty in the risk estimates, and the relative contributions of the input variables to that uncertainty.) The qualitative approach has the advantages of being convenient and inexpensive, and providing single point estimates of risk that are easily understood. Moreover, a qualitative approach can be used to highlight and at least partially address uncertainties not amenable to quantification. To this end, virtually all uncertainty assessments will be qualitative to a degree. The disadvantages of the qualitative approach are that the information provided may not be sufficient to support decision-making about risk management or about the need for additional data gathering, and the point estimates of risk may be misinterpreted as being highly accurate and certain, when in fact they are not.

**Multi-Scenario Approaches and Limited Sensitivity Analysis.** Early efforts at quantitative uncertainty and variability assessment often employed limited sensitivity analysis of key variables. In this approach, plausible values of what are believed to be key variables (high-end, central tendency, low-end, etc.) are combined to develop multiple point estimates of risk, and the range of the estimates is used as an indication of the overall uncertainty of the analysis. While in principle this approach may provide useful information about the magnitude of, and the major contributing factors to the overall uncertainty, in practice sensitivity analyses are often limited to only those variables for which data are available (which is true of all quantitative treatments of uncertainty). Also, the combinations of variable values that are used to derive the various risk estimates may not be physically plausible. Resource costs for this approach are generally relatively low, but again the information provided may not be sufficient to guide decision-makers.

**Systematic Sensitivity Analysis.** When sufficient data are available, the contributions of individual variables to risk estimates may be evaluated systematically by a related set of methods that fall under the general heading of sensitivity analysis. Such approaches can use statistical techniques such as correlation analysis, multiple regression, and response surface evaluation (Morgan and Henrion 1990). These methods generally involve calculating risks using a large number of plausible combinations of plausible values of all of the input variables, and using statistical methods to elucidate the impacts of each variable and combination of variables on the risk assessment results. These approaches have the advantage that they usually will provide a great deal of information on uncertainty and variability impacts. This is offset by the disadvantage that, with even a relatively small number of input variables, the methods rapidly become very complex, cumbersome, and resource-intensive, and the results can be very difficult to interpret.

**Monte Carlo Simulation and Related Probabilistic Methods.** Another set of methods commonly employed to evaluate the effects of uncertainty and variability in risk assessments are explicitly probabilistic in nature. They take as their inputs probability distribution functions<sup>3</sup> (rather than discrete single values) of the major variables in the risk assessment, and use Monte Carlo or other simulation methods to combine them into a probability distribution of risks. These methods were not widely employed in health risk assessment until rather recently, when inexpensive PC-based simulation packages became widely available. These methods have the advantage that they are theoretically rigorous; that is, if the simulation is performed correctly, the output risk distribution exactly reflects the multiple contributions of all of the input variables. Monte Carlo methods are also easily amenable to sensitivity analysis of the impacts of specific variables on risk outcomes. Recently, methods have been developed that can simultaneously evaluate the separate contributions of variability and uncertainty in individual variables to overall uncertainties in risk estimates, and that can take into account the effects of correlations among input variable values. Simulation methods can be technically complex, however, and are very data-intensive, and the

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<sup>3</sup> A probability distribution function can be thought of as a continuous graph of the probability that a variable will take a specific value within a specific range. Often, a cumulative probability distribution function is used, which graphs the probability that a variable value will be less than a given value.

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results depend strongly on the availability of information or the resources to gather information. Finally, the outputs of simulation models may be difficult to interpret for stakeholders and risk managers accustomed to discrete risk estimates. At the present time, simulation modeling can rarely be used to capture all sources of variability and uncertainty quantitatively.

### **Strategy for Considering Uncertainty in Residual Risk Analyses**

Each of the general approaches to uncertainty and variability assessment have specific advantages and disadvantages, and different approaches may be appropriate in different situations, depending on the nature of the assessment being undertaken. In the assessment of residual risks, it is likely that no single method will be appropriate for all assessments. Depending on the importance (magnitude) of risks for given source categories, the availability of data, and the costs of reducing risks, more or less detailed uncertainty analysis may be justified. The 1992 Exposure Assessment Guidelines recommend a “tiered approach” to risk assessment, which will likely be adapted to uncertainty evaluation as well. Simple, conservative, generic approaches are stressed early in the process of the assessment when specific chemicals, exposure pathways, and regulatory scenarios are being screened to identify which are most likely to be associated with the highest risks. Then, more refined models, supported by more specific data, are applied until sufficient information about risks is obtained to satisfy the needs of the decision in question.

As for the residual risk assessments themselves, EPA’s general policy for uncertainty evaluation in residual risk assessments will rely on simple screening-level methods where there is relatively little evidence that risks are likely to be significant, progressing to higher degrees of refinement as needed to support management decisions addressing higher residual risks. As discussed further below, it is likely that the Agency’s main concern in uncertainty evaluations for residual risk assessments will be ascertaining, with an acceptable degree of certainty, that risks do not exceed levels of concern, rather than detailed elucidation of the contributions of specific variables to the overall uncertainty of the risk estimates. For this purpose, the simple multi-scenario approach may be sufficient to provide the needed degree of assurance, and more complex methods may not be needed. Only where risks appear to be significant, and where there is a significant degree of uncertainty (or variability) associated with specific variables, are more detailed methods likely to be appropriate.

## **Uncertainty and the Management of Residual Risks**

Evaluation of uncertainty used to be regarded primarily as a statistical problem. The developers of more sophisticated methods for uncertainty and variability analysis saw their approaches as being self-evidently superior to existing methods, and concerned themselves primarily with perfecting the technical aspects of uncertainty evaluation. Significantly less effort was devoted to the development of methods for presenting the results of the analyses, and their application in risk management decisions. (It was again assumed that this was primarily a technical problem that could be addressed by formal decision analysis or related methods.) As more experience has been gained in the application of quantitative uncertainty evaluation to risk management decisions, it has become clear that fundamental issues of utility and intelligibility need to be carefully addressed as a fundamental part of the assessment process, and need to be factored into such analyses from their earliest stages. Particularly, as the Federal government pursues its goals of expanded stakeholder involvement in risk management decisions (CRARM 1997a,b), a premium is being placed, as it should be, on providing information that is useful and intelligible to non-technical audiences.

The utility of information is of foremost importance. The results of any assessment of uncertainty evaluation should answer questions relevant to the specific decision being made. As noted in Section 4.1.1, the Agency is required to evaluate the “public health significance” of residual risks. This level clearly has a probabilistic component; e.g., how certain does the Agency need to be that a risk is or is not “significant”? Is there some intermediate combination of risk and uncertainty that indicates the need for more data gathering, rather than immediate management? How can uncertain risks be compared and prioritized? The answers to these questions depend not only on the nature and magnitude of the risks being evaluated, but also on the specific control options available and their economic impacts. Especially in light of recent Congressional calls for more detailed analysis of regulatory impacts through cost-benefit and related methods (for example, U.S. Senate 1997), the Agency must develop consistent approaches to defining the need for uncertainty evaluation for residual risk management and other major rules. Such policies will likely not specify specific numerical standards, but must provide a consistent framework for regulatory analysis. This guidance must then be made operational appropriately in residual risk assessment.

The complexity of uncertainty evaluation, and particularly of probabilistic methods, imposes a real barrier to understanding (and thus, to utility). Most stakeholders are accustomed primarily to point estimates of risk and simple dichotomous decision rules. (If risk is above a certain level, take a certain action. If not, take another action.) In contrast, it may not be intuitively obvious, even to relatively sophisticated audiences, how to relate the outputs of quantitative uncertainty evaluation to a particular decision. At best, quantitative uncertainty evaluation adds an additional level of complexity (a measure of confidence, along with a risk estimate), to decision-making. In complex analyses, important aspects of the regulatory decision may rest on relatively subtle statistical distinctions (e.g., between a 95th percentile risk estimate and an upper 95th percentile confidence limit on a risk estimate), and the challenges in presenting

such information can be formidable. In its recent guidance, the Agency has begun to define concrete approaches to the presentation of risk and uncertainty information to decision-makers and stakeholders. These efforts will need to be continued and elaborated in the course of the Agency's residual risk assessments.

#### **4.2.4 Negative Health or Environmental Consequences**

This section addresses the CAA section 112(f)(1)(C) requirement to investigate and report on "...any negative health or environmental consequences to the community of efforts to reduce such [residual] risks." Pollution control technologies targeted at a single pollutant (e.g., a specific HAP) and single medium (e.g., air), especially conventional end-of-the-pipe treatment technologies, can inadvertently transfer pollutants and risks to different media, different locations, and different receptors, and can unintentionally create new and different risks in the process of controlling the targeted risk. Few control technologies, when viewed from a holistic, multimedia, life cycle perspective, are without health and environmental risks of their own. In the context of HAP residual risk, for example, a technology that removes a HAP from an air emission stream can produce contaminated water and/or solid waste, can require additional energy (which consumes resources and produces other pollutants), and in some cases may create new safety risks, especially for workers.

The EPA recognizes the possibility of creating or transferring risks as an unintended byproduct of actions that may be taken to reduce residual risks of HAPs. Thus, EPA intends, as part of the section 112(f) standard-setting process, to identify and estimate significant negative health and environmental consequences and consider the risk-risk tradeoffs associated with any standards established under the residual risk program. One of EPA's primary goals is to ensure that measures taken to reduce risk under section 112(f) authorities do not create other risk problems.

A key step in the residual risk process for HAP source categories determined to need additional risk reduction beyond the MACT standards in place will be the development and analysis of a range of risk management options. Ultimately, a risk management approach will be selected for the source category and a standard developed under section 112(f) to reduce risks to acceptable levels. As part of the analysis of risk management options – which will include evaluation of the effectiveness, reliability, emission and risk reduction, and cost of each option – the potential negative health and environmental consequences will be identified and analyzed as well. In other words, EPA will consider the broad range of positive and negative impacts of each risk management option under consideration, rather than focusing simply on one criterion, such as control efficiency or cost. Information describing and, where practicable, quantifying potential negative consequences will be presented along with the other critical information to decision-makers responsible for selecting the risk management strategy.

In contrast to conventional air pollutant removal and treatment technologies, many pollution prevention approaches to reducing residual risks have fewer negative health and environmental consequences. This is primarily because pollution prevention approaches eliminate pollutants (and thus emissions) at the front end of a process rather than attempting to treat and dispose of them at some downstream step of the process. Thus, EPA will ensure that pollution prevention approaches are identified as risk management options and seriously considered in the standard-setting process. There will be a strong preference for selecting feasible pollution prevention approaches to reduce the residual risks of HAPs, in large part because they generally have fewer negative health and environmental consequences than other options.

#### **4.3 Section 112(f)(1)(D): Legislative Recommendations**

Section 112(f)(1)(D) gives EPA the opportunity to make “recommendations as to legislation regarding such remaining risk” that may be identified during the analysis for residual risk. The EPA has interpreted this to mean that if an unacceptable residual risk were identified and no current authorities under the 1990 CAA Amendments were determined to be adequate to reduce that risk, then EPA would have the authority to propose an approach that would ensure that risk reductions would occur. If Congress did not act on any proposed recommendation or if the Agency did not propose any legislative recommendations, then EPA has the authority to set additional standards as described in section 112 (f)(2).

The EPA is not proposing any legislative recommendations to Congress in this report. The legislative strategy embodied in the 1990 CAA Amendments adequately maintains the goal of protecting the public health and the environment and provides a complete strategy for dealing with a variety of risk problems. The strategy recognizes that not all problems are national problems or have a single solution. National emission standards will be promulgated to decrease the emissions of as many HAPs as possible from major sources. The authority is also provided to look at smaller scale problems such as the urban environment or the deposition of HAPs to water bodies in order to address specific concerns, to focus or prioritize efforts to meet specific needs such as a concern for a class of toxic and persistent HAPs, and to allow for partnerships among EPA, States, and local programs in order to address problems specific to these regional and local environments. Congress developed a strategy that, when taken as a whole, provides EPA with the flexibility to identify and deal with a wide range of air toxics problems. It should be kept in mind, however, that until the actual nature and scope of the potential residual risks are known, the adequacy of the CAA strategy can only be assumed.

Residual risk will play a major role as EPA moves into the risk-based phase of the CAA strategy. Using information gathered from a variety of sources including Congressionally mandated studies, the residual risk program will provide part of the “safety net” that will insure

that the public and the environment will be protected. The following chapter describes this program's strategy in more detail.

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## **5. The Residual Risk Strategy**

The remainder of section 112(f) – sections 112(f)(2) through (6) – provides guidance on what EPA is to do if Congress does not act on any legislative recommendations in the section 112(f)(1) report. In section 112(f)(2), Congress requires EPA to promulgate standards in order to provide an “ample margin of safety” to protect the public health and to prevent, taking into account costs and other relevant factors, an “adverse environmental effect.” Given that this report does not present any legislative recommendations, this chapter describes EPA’s overall goals and strategy for conducting residual risk analyses in response to the requirements of sections 112(f)(2) through (6).

### **5.1 Legislative Context for the Residual Risk Strategy**

#### **5.1.1 The Context for the Analyses**

Congress has defined the context for residual risk standards to be the list of source categories or their subcategories that have been subjected to emission standards under section 112(d) of the CAA. On December 3, 1993, EPA established the promulgation schedule for technology-based (MACT) emission standards for 174 listed source categories (EPA 1993e). The source categories were divided into four groups, or bins, based on their expected promulgation date: 1992, 1994, 1997, and 2000 (also referred to as 2-year, 4-year, 7-year, and 10-year bins). MACT is intended to identify and control air emissions from those major sources that emit any of the HAPs listed pursuant to section 112(b) of the CAA. For existing sources, the minimum level of emissions reduction to be achieved is determined by establishing the current level of control of the best controlled 12 percent for each source type and establishing a “floor level” that is the average of the 12 percent level. MACT emission reductions are based on source and technology analyses and do not consider risks presented by potential HAP exposures.

However, Congress intended risks to eventually be considered, as evidenced by the fact that most of the CAA-mandated air toxics programs other than MACT involve risk analyses and strategies to reduce risk to the public and environment. Congress stated in section 112(f)(2) that if a single facility within a source category subject to standards under the MACT program has an estimated lifetime excess cancer risk to the individual most exposed of greater than one in a million, then the Administrator shall promulgate new standards to protect the public health. EPA does not consider the one in a million individual cancer risk level as a “brightline” cutoff for making risk reduction decisions, but rather as a trigger point to conduct more refined risk assessments. This interpretation is supported by the guidance provided in the September 14, 1989 *Federal Register* notice promulgating national emissions standards for benzene (i.e., the benzene NESHAP), which is cited in section 112(f) (see Section 2.1 for more discussion of the benzene NESHAP, and Appendix B for excerpts from the preamble to the final regulation). EPA will continue to use this guidance for making final risk management decisions under section 112(f) for carcinogens rather than adopting any single “brightline.”

Residual risk is one of the air toxics programs that begins to shift the emphasis toward the receptors being exposed. While the source category as context defines the range or scope of the data that will be required for performing the residual risk analyses, the receptor (i.e., the human populations or the particular environments affected) defines the context for the characterization of the risk estimated from the analyses. The HAPs emitted, the routes of exposure, and the nature of the populations or environments being exposed become very important to the risk assessment outcome.

### **5.1.2 Compliance Schedule and Effective Date**

According to section 112(f)(2), residual risk standards must be promulgated within eight years of the promulgation date of the MACT standard for that category unless the source category MACT was scheduled for promulgation within the first two years after the date of enactment of the 1990 CAA. In the latter case, residual risk standards must be promulgated within nine years. Therefore, for purposes of any residual risk standards, the eight-year limit applies to all source categories listed in the 4-, 7-, and 10-year bins, and the nine-year limit applies to categories listed in the 2-year bin, regardless of the actual promulgation date. This means that the earliest standards promulgated under the residual risk program are due to be finalized in the year 2002 (earliest MACT promulgation for a category in the 2-year bin was 1993). Appendix C contains tables of the source category MACT standards, organized according to their promulgation schedule, and the actual promulgation dates of those that have been issued.

Section 112(f)(3) establishes that standards will become effective upon promulgation, although section 112(f)(4) provides existing sources subject to residual risk standards a 90-day time period after promulgation to comply, unless the Administrator grants a compliance waiver of up to two years. The Administrator must assure that during the waiver period “the health of persons will be protected from imminent endangerment.”

### **5.1.3 Area Sources (CAA Section 112(f)(5))**

Area sources are defined as sources that have the potential to emit less than 10 tons/year of a single HAP or 25 tons/years of HAPs in aggregate. Section 112(f)(5) stipulates that the Administrator shall not be required to conduct a residual risk review of any category or subcategories of listed area sources for which an emission standard, referred to as Generally Available Control Technology (GACT), has been promulgated under section 112(d)(5). The EPA interprets this statutory language to mean that any area source for which the emission standard is based on MACT will be included in the residual risk analyses according to its specific schedule of promulgation, but an area source for which GACT was the basis of the standard will be reviewed under the residual risk program only if deemed necessary by EPA. Area sources to which MACT has been applied are identified in Appendix C.

#### **5.1.4 Unique Chemical Substances (CAA Section 112(f)(6))**

There are 17 HAPs listed under section 112(b) that are not specific individual compounds and for which no CAS numbers are given (see **Exhibit 15**). Eleven of these are classes of metal compound HAPs, and the rest cover a variety of other HAP classes. Congress has directed in section 112(f)(6) that in setting residual risk standards applicable to sources that emit any of these HAPs, the Administrator should consider information on the HAP that is actually emitted. Each of these HAP classes may contain hundreds of individual compounds for which there may be very limited or no toxicity, emissions, or other risk-related data. The EPA has defaulted to relying on data from unspiciated HAPs in this category of "non-CAS number HAPs" as the basis for evaluating risks, or has used data for one member of a class as a surrogate for other members of the class that have data gaps. In the absence of toxicity, emissions, and other risk-related information about the specific "non-CAS number HAPs" that may be emitted by a source under study, EPA will continue to use information that is available on any of the constituents, including the elemental compounds, when making residual risk determinations. Where substance-specific data are available, EPA will use such data when making residual risk determinations.

An additional requirement of section 112(f)(6) is that any direct transformation byproducts resulting from the emissions of any of these classes of HAPs should be the basis for setting standards.

### **5.2 Residual Risk Analysis Goals**

Congress' goals for the residual risk program, as expressed in section 112(f)(2), are to: (1) assess any risks remaining after MACT standard compliance; (2) determine if additional emission reductions are necessary and, if so, for which source categories; (3) set a standard that protects the public with an "ample margin of safety;" and (4) set a more stringent standard if necessary, taking into account cost, energy, safety, and other relevant factors, to prevent an adverse environmental effect.

Using the guidance provided in section 112(f) and the risk assessment process described in Chapter 3, EPA will evaluate all of the source categories for which MACT standards are promulgated under section 112(d). The MACT program has succeeded in achieving substantial emissions reductions across many HAPs and industries. In doing so, it has leveled the emissions playing field within industry types and has reduced risks as well. The residual risk strategy is intended to provide the Agency flexibility in its decisions while ensuring that the public and environmental health is protected. EPA's goals also include continuing the partnership with State/local programs in the sharing of data and expertise, and including all groups who may be affected by residual risk decisions (e.g., industry, environmental groups) as part of the process, beginning with the analysis.

**EXHIBIT 15**  
**17 HAP CLASSES LISTED UNDER CAA SECTION 112(b)**

Antimony Compounds
Arsenic Compounds (inorganic including arsine)
Beryllium Compounds
Cadmium Compounds
Chromium Compounds
Cobalt Compounds
Coke Oven Emissions
Cyanide Compounds <sup>1</sup>
Glycol Ethers <sup>2</sup>
Lead Compounds
Manganese Compounds
Mercury Compounds
Fine Mineral Fibers <sup>3</sup>
Nickel Compounds
Polycyclic Organic Matter <sup>4</sup>
Radionuclides (including radon) <sup>5</sup>
Selenium Compounds

<sup>1</sup> X'CN where X = H' or any other group where a formal dissociation may occur. For example, KCN or Ca(CN)<sub>2</sub>.

<sup>2</sup> Includes moni-and di-ethers of ethylene glycol, diethylene glycol, and triethylene glycol R-(OCH<sub>2</sub>CH)<sub>n</sub>-OR' where:  
n = 1, 2, or 3

R = alkyl or aryl groups

R' = R, H, or groups which, when removed, yield glycol ethers with the structure:

R-(OCH<sub>2</sub>CH)<sub>n</sub>-OH.

Polymers are excluded from the glycol category.

<sup>3</sup> Includes mineral fiber emissions from facilities manufacturing or processing glass, rock, or slag fibers (or other mineral derived fibers) of average diameter 1 micrometer or less.

<sup>4</sup> Includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100°C.

<sup>5</sup> A type of atom which spontaneously undergoes radioactive decay.

### **5.3 Residual Risk Strategy**

Using the context provided by Congress in section 112(f) and the methodologies, data, and assessment process for air toxics described in more detail in previous sections of this report, EPA has developed a residual risk strategy. The strategy for residual risk analysis may be described in several steps: identifying management goals that reflect the legal requirements, problem formulation, data collection, exposure and toxicity assessment, risk characterization, and risk management/risk reduction. **Exhibit 16** presents a flowchart of the general residual risk strategy. In short, the strategy calls for an iterative, tiered assessment of the risks to humans and ecological receptors through both direct and multipathway exposures to HAPs, leading ultimately to a decision on whether additional emission reductions are needed for individual source categories. This type of iterative, tiered approach is consistent with the NRC (NRC 1994) and Risk Commission (CRARM 1997a,b) reports written pursuant to the 1990 CAA Amendments. A more detailed decision framework for ecological risk, which is derived both from this overall residual risk strategy and from EPA's ecological risk framework and guidelines, is presented in Section 5.4.

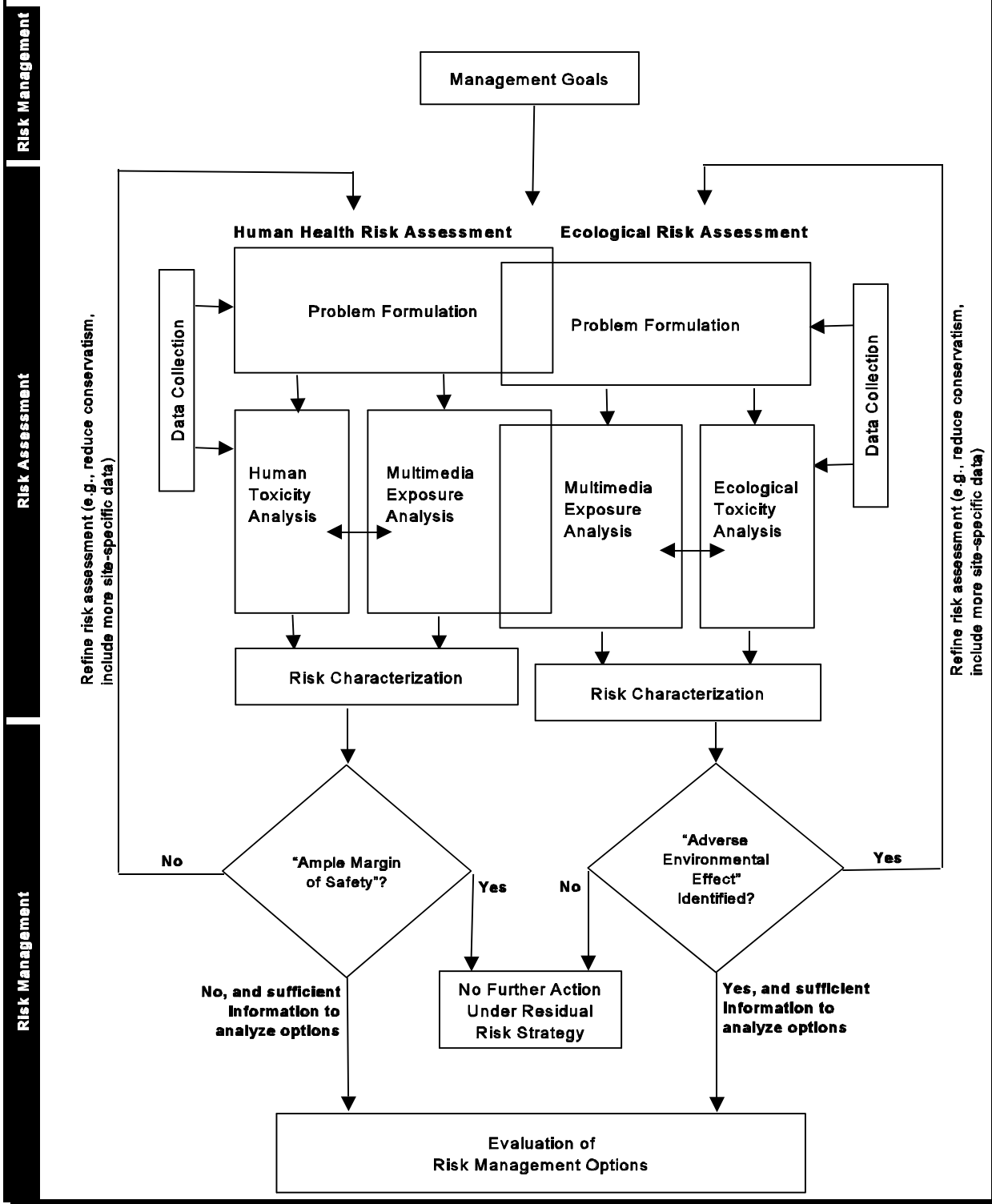
The first component of the residual risk strategy is that EPA state its risk management goals, which are identified at a broad level in the CAA legislation:

- to achieve a level of emissions that ensures that the public health is protected with an “ample margin of safety;” and
- to ensure, taking into account cost, energy, safety, and other relevant factors, that residual emissions do not result in “an adverse environmental effect.”

EPA may decide to translate those legislative objectives into more specific management goals. Those management goals help direct the problem formulation phase of both the human health and ecological risk assessments.

For both the human health and ecological risk assessments, the basic premise of the tiered approach is that early tiers are generally screening in nature, which means that they are designed to be relatively simple, inexpensive, and quick, using existing data, defined decision criteria, and models with simplifying assumptions as inputs. Later tiers refine some or many aspects of the analysis, depending on which influence risk most and are most uncertain. Later tiers require more resources, but the results are less uncertain and less conservative (i.e., less likely to overestimate risk). While the strategy is represented generally as having two tiers (screening and refined), multiple analyses might be performed in an iterative fashion within one or both tiers. The key point is that successive analyses of increasing complexity (and resource requirements) will be performed in a manner EPA determines is cost-effective for a given source category.

**EXHIBIT 16  
OVERVIEW OF RESIDUAL RISK STRATEGY  
TIERED (ITERATIVE) APPROACH**



In using this approach, EPA will follow the recommendation of the NRC (1994, p. 9) which stated “EPA should use bounding estimates for screening assessments to determine whether further levels of analysis are necessary. For further analysis, the committee supports EPA’s development of distributions of exposures based on actual measurements, results from modeling, or both.” The EPA believes that the first tier being evaluated for use in screening level assessments does, in most cases, produce bounding estimates. However, if this tier is so conservative that source categories will not be screened out for further consideration under the residual risk program, additional tiers which use more reasonable assumptions will be evaluated and used. When a decision is made to do a more refined analysis, the exposure assessment will use distributions of exposures and follow the process that was discussed in Section 3.1.4 of this Report.

As shown in Exhibit 16, each tier of the human health and ecological risk assessments is organized into three phases: (1) the problem formulation phase, in which the context and scope of the assessments are specified; (2) the analysis phase, in which the HAPs’ toxicity and exposure to humans or ecological receptors are evaluated; and (3) the risk characterization phase, in which the toxicity and exposure analyses are integrated to assess the nature, magnitude, and uncertainty of any risks. Also as illustrated in Exhibit 15, the problem formulation and analysis phases of the human health and ecological risk assessments will partially “overlap” in that certain pathways of concern for humans (e.g., consumption of contaminated fish) might also be pathways of concern for ecological receptors (e.g., fish-eating wildlife).

Following the risk characterization phase of each tier of the assessment, a risk management/risk reduction decision is made. If an “ample margin of safety” has been met for the human health risks and no “adverse environmental effect” has been identified, then no further action is required, and the results of the risk assessment should be documented. If an ample margin of safety is not met (and/or environmental effects are possible) and if sufficient information is available to evaluate management options considering risks, costs, economic impacts, feasibility, energy, safety, and other relevant factors, the risk assessment is complete (i.e., no additional tiers are needed), and options for reducing risk should be analyzed. If the information from the risk characterization is insufficient to fully evaluate risk management options, the residual risk assessment should proceed to a more refined analysis.

The Exhibit 16 flowchart applies to the analysis and decision-making for an individual source category. Priority-setting among the 174 source categories to be reviewed – that is, determining the order in which residual risk assessments will be conducted – also is a critical part of the strategy. EPA intends to set priorities based on a number of considerations, including the actual MACT promulgation dates for source categories (which starts the clock on the statutory time period for residual risk determinations) and any available information bearing on the level of residual risks attributable to various source categories. To the extent possible, based on the available data, EPA will set priorities so that it achieves the largest possible risk reductions first. Priority-setting also will be iterative; priorities are likely to be revised during the course of the

residual risk program as new information becomes available and analyses are performed on various source categories.

### **5.3.1 Problem Formulation**

Residual risk analysis for a given source category will begin by describing, as completely as possible, the context and scope of the problem being evaluated. As many data as are readily available will be used at this stage of the assessment, and stakeholders with concerns or interest about this category may be encouraged to provide input. Information from State, local, and Tribal entities may help the planning process by pointing out source categories or HAPs of concern, or by identifying issues to consider. It would be at this stage that key decisions about the HAPs of concern would be made. For example, do the HAPs being emitted raise concerns for the need to do multipathway human health or ecological assessments as well as inhalation assessments? What are the endpoints of concern, and what populations may be most affected by the HAPs being emitted? These evaluations may be largely at a qualitative level, but they will inform the design of the analysis to follow, both the screening and refined levels.

As pointed out above, the timing of the MACT promulgation schedule will require some prioritization of work to occur. A number of source categories may be scheduled for analyses during the same time period. The problem formulation phase will help to prioritize which source categories need earlier attention. It also will help determine what data are needed to support certain decisions and whether those data are available.

Designing the risk assessments during problem formulation involves the following main activities:

- Characterize key sources of HAP release;
- Characterize environmental behavior of HAPs and determine for which, if any, multipathway analyses might be required;
- Identify receptors that are potentially at risk;
- Select assessment endpoints; and
- Identify exposure pathways of concern.

### **5.3.2 Data Collection**

Many types of data from a wide variety of data sources are needed to assess the residual risks of source categories. Data collection is expected to be iterative and to occur throughout the residual risk assessment process, beginning with problem formulation. Some data collection is needed even before any screening analyses are begun on individual source categories, to serve as a basis for setting priorities and ordering the source categories for residual risk assessment. Then, in conjunction with problem formulation, some data collection is needed to provide inputs for the screening-level risk assessment. Because the screening assessment is intended to be based on readily available data, this step generally will be gathering and organizing the existing data (e.g.,

health and environmental effects of HAPs, post-MACT source emission rates for HAPs), generally from EPA sources (e.g., MACT rulemaking docket, MACT data base) and State and local air toxics agencies.

For source categories that proceed from screening to refined risk assessments, additional data collection will be required, with a greater emphasis on site-specific data for affected facilities. In some cases, this data collection effort may be relatively extensive, although it should be able to be focused – based on the results of the screening assessment – on the HAPs, types of effects (i.e., endpoints), sources, locations, exposure pathways, and receptors of most concern. Data collection to support the refined assessment may involve more detail about data elements used in the screening assessment (e.g., HAP emission rates, source characteristics) as well as information about additional data elements (e.g., exposed populations and subpopulations, epidemiology and disease registry information, actual ecosystems and endangered and threatened species that might be exposed). This data collection step is also more likely to include collection of data from industry sources and possibly other stakeholders, in addition to more extensive data collection from State and local agencies.

The sources of this additional information for the refined assessment will vary. It is assumed that State, local, and EPA Regional offices should have information that is more site-specific, especially about which facilities are subject to a particular MACT rule, which have applied for operating permits, and which are in compliance at a particular time. Other facility-specific information that is needed to do the more detailed exposure and risk analysis may have to be obtained from the information request mechanisms that were used to gather data for the MACT process. Other information needed may come from existing data bases, such as U.S. Census data, geographic information systems (GIS), or other types of data bases that may provide needed inputs for modeling. EPA may also work together with industry to obtain needed data, or may make use of mechanisms such as Information Collection Requests (ICRs) or CAA section 114 letters to obtain needed information.

The data available will in part determine whether an analysis is done on specific facilities in a source category or on model plants of the type developed during MACT rule development. EPA anticipates that the amount of information available about facilities within a source category may be more extensive after the Agency promulgates a MACT standard versus what was known during MACT rule development. Some of the anticipated information would be knowledge of the HAPs being emitted, the regulatory level or estimated emission reductions for these HAPs, the locations of the facilities subject to a MACT rule, and whether a specific facility is in compliance with the rule. This type of information could narrow the scope of the refined analysis to those facilities that appear most likely to be a residual risk concern.

### **5.3.3 Exposure and Toxicity Assessments and Risk Characterization**

EPA's overall approach to air toxics risk assessment was described in Chapters 3 and 4 of this report. The residual risk strategy incorporates the methods and data discussed in those chapters. The general framework for evaluation of risks remaining after application of MACT is an iterative, tiered system of exposure and effects assessment and risk characterization.

As illustrated in Exhibit 16, the strategy consists of different tiers of evaluation that may be broadly described as screening-level analyses and more refined evaluations. These different levels of analyses are designed to allow the assessment to proceed in a timely way without unnecessary investment in data collection. A conservative screening-level risk assessment, which is designed to be relatively simple, inexpensive, and quick, uses existing data, defined decision criteria, and models with simplifying assumptions as inputs. For example, data on exposed populations or the actual ecosystems surrounding specific facilities generally would not be used in a screening assessment, but would be reserved for the refined assessment. A basic premise of the tiered approach is that simple approaches are used first to determine whether or not the source of emissions poses a potentially unacceptable risk. More refined tiers might be conducted for those aspects of the risk assessment that are most important to the results or are responsible for the greatest uncertainty in the results. The more refined tiers require more resources, but should be more certain and less conservative (i.e., less likely to be overestimates of risk). Where risks are obvious, more refined analyses might be implemented at the start.

Screening-level analyses will be applied to assess both human health and ecological risks, and will be used to assess both direct and multipathway exposures. When the screening assessments are complete, EPA will assemble the information it has collected, as well as the results of any screening analyses, to prepare a characterization of the source category that would describe any potential public or environmental health concerns. This information may include both quantitative and qualitative data and results. The screening assessment results will be used to eliminate low-risk source categories, prioritize the remaining source categories for refined assessments, and also to focus the refined assessments so that they can be done more efficiently.

While the screening analyses can serve as a basis for a decision to pursue additional analyses or to eliminate low-risk source categories from further consideration under section 112(f), they are not adequate to serve as a basis for establishing additional emission reduction requirements under section 112(f). Refined assessments will be used as the basis for deciding whether additional emission reductions are needed and, if so, for determining what level of reductions are appropriate. Considerable professional judgment is required to carry out and interpret a more refined residual risk assessment, and the steps taken and approaches used may vary from one source to the next, even within the same source category. As noted earlier, continued refinements might be necessary for some or all components of the analyses.

Consistent with recent recommendations of the Presidential/Congressional Commission on Risk Assessment and Risk Management (CRARM, 1997a,b) and the National Research Council

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(NRC 1996), an additional component of the problem formulation phase for the more refined analyses will be the involvement of affected groups in the process. EPA will make its information available to State and local public health and air toxics agencies, affected industries, and concerned Tribal and public interest groups, and will take other steps to facilitate meaningful stakeholder participation. Stakeholder involvement adds another dimension by allowing affected parties to have input and to be given the opportunity to understand the views of other participants. This will be especially critical when reviewing the results of the screening-level analyses. The results of a screening-level modeling exercise may not provide a sufficiently complete picture of whether existing emissions are acceptable. EPA will rely on feedback from stakeholders whose concerns may extend beyond the technical capabilities of modeling to better discern the complete problem. For example, while the scope of the residual risk analyses will be national, it is possible that local, State, or regional level problems would only be brought to light by groups at that level. Stakeholder involvement may not be the same for all analyses. The level of stakeholder involvement may be driven by the extent of the analyses and the expected impacts of decisions that will result from the analyses.

#### **5.3.4 Risk Management/Risk Reduction Decisions**

In addition to the results of the risk analysis/characterization based on human health and environmental data, the risk manager is also required by CAA section 112(f) to consider other factors before recommending the establishment of additional risk standards. In determining whether further regulation is warranted in order to protect public health with an ample margin of safety and/or to prevent an adverse environmental effect, the risk manager will evaluate the level of risk and the risk reduction achievable against costs, feasibility, and other factors and, in the case of environmental risks, against costs, energy, safety, and other relevant factors.

The key risk management decision points within the strategy occur after the risk characterization step in each tier of the risk assessments, at the conclusion of the screening assessment and, where applicable, the conclusion of the refined assessment. The outcome of the decision following the screening assessment has two possibilities: (1) "no further concern **at this time**," or (2) "more detailed data collection and/or analysis is warranted." To consider a source category to be of no further concern under the residual risk program, all of the health criteria ("ample margin of safety") and environmental criteria (no "adverse environmental effect") would need to be satisfied. The health criteria are discussed in Section 4.1.1, and the cancer risk criteria are summarized in **Exhibit 17** (same as Exhibit 14 in Chapter 4). The environmental criteria are discussed in Section 5.4. Where the available information is too limited to make a "no further concern" determination, those components of the source category responsible for the uncertainty would be subject to more data collection and more refined analysis. If the decision is made not to continue the analysis of a source category at the conclusion of the screening assessment, then the information supporting that decision would be made available to stakeholders. A decision to

**EXHIBIT 17**  
**SUMMARY OF CRITERIA FOR EVALUATING PUBLIC HEALTH SIGNIFICANCE**  
**FOR CARCINOGENS**

Effect Type	Screening Level <sup>1</sup>	Refined <sup>2</sup>
Cancer	<ul style="list-style-type: none"><li>▶ Upper-end individual risk <math>&lt;10^{-6}</math> generally meets ample margin of safety; <math>\geq 10^{-6}</math> generally leads to refined analysis</li><li>▶ Assume additivity for all HAPs</li><li>▶ Confidence in toxicity values not necessarily considered</li><li>▶ Size and nature of potentially exposed population not necessarily considered</li></ul>	<ul style="list-style-type: none"><li>▶ Upper-end individual risk <math>&lt;10^{-6}</math> generally meets ample margin of safety; <math>\geq 10^{-4}</math> generally does not meet ample margin of safety</li><li>▶ Upper-end individual risk between <math>10^{-6}</math> and <math>10^{-4}</math> may meet ample margin of safety, depending on confidence in the risk estimate, population size, presence of sensitive subpopulations at various risk levels, and other factors</li><li>▶ Assume additivity for all HAPs</li></ul>

<sup>1</sup> Screening based on upper-end estimated HAP exposure at the location of either the MIR or MEI. All available toxicity values will be considered.

<sup>2</sup> Refined assessment based on more detailed and site-specific, and less conservative, estimated HAP exposures at the MIR location and throughout the spatial area of impact. EPA consensus toxicity values, or equivalent, are required.

recommend a source category for more refined analysis would require that only one of the criteria be exceeded.

If a decision is made that additional emission reductions should be considered, then EPA will establish standards that protect public health with an ample margin of safety (considering costs, feasibility, and other factors in addition to health risks) and, where necessary, that prevent an adverse environmental effect (considering cost, energy, safety, and other relevant factors in addition to environmental risks). EPA will use its CAA section 112(f)(2) residual risk authority to set national standards but will focus the applicability of any standards only to those portions of a source category that fail to meet acceptable risk levels.

Thus, an important component of this process will be the establishment of interactive discussions with all parties involved. The stakeholders in this case are State and local public health and air toxics agencies, the affected industry, and concerned Tribal and public interest groups. The purpose of these interactions would be to discuss the results of the risk assessments, to determine the nature and the scope of the potential risks, to hear concerns and perceptions about the level of risk, to discuss the next steps in the process, and to discuss the options available to reduce risk if necessary. The opening of a stakeholder dialogue, consistent with legal limitations such as the Federal Advisory Committee Act, provides the opportunity for all groups to be involved early in the process and for the implementation of a rational risk reduction strategy that proceeds from mutual understanding rather than a one-sided argument.

### **5.3.5 Comparison of EPA's Strategy with CRARM Recommendations**

In formulating its strategy for assessing residual risks under the CAA, EPA has conformed to many of the specific recommendations articulated by CRARM in their 1997 final report (CRARM 1997a,b). EPA's overall consistency with the tiered approach advocated by the Commission (see Exhibit 3) is evident throughout this Report in the methods and strategies described (see, for example, Exhibit 16). In addition, five specific recommendations of the Commission (see Section 2.3.2) are listed here along with a short explanation of how EPA is fulfilling each.

- *Characterize and articulate the scope of the national, regional, and local air toxics problems and their public health and environmental contexts.*

EPA is in the process of defining an Air Toxics Strategy that will assess what we know about these problems and will identify how the provisions in section 112 can best address them. In addition, EPA is characterizing more specific issues as directed by Congress in the CAA and through Reports to Congress and programs addressing the Great Waters, Mercury, and the Urban Air Toxics Problem. The residual risk program, through which post-MACT risks from industrial source categories are being assessed, is also an element in EPA's characterization of the air toxics problem.

- *Use available data and default assumptions to perform screening level risk assessments to identify sources with the highest apparent risks.*

This is the underlying strategy of EPA's residual risk approach described throughout this report and illustrated in the flow chart in Exhibit 16. The flow chart is an adaptation of the approach proposed by the Commission in their 1997 final report.

- *Conduct more detailed assessments of sources and facilities with the highest risks, providing guidance and incentives to regulated parties to either conduct these risk assessments or reduce emissions to below screening thresholds.*

EPA is currently evaluating the potential for both EPA and regulated parties to carry out detailed risk assessments, when appropriate based on screening assessment results, using the methods described in detail in Chapter 3 of this Report. EPA will develop guidance for such assessments as necessary.

EPA will consider incentive to industry to reduce residual risks, as described in Section 4.1.2.

- *At facilities that have incremental lifetime upper-bound cancer risks greater than one in 100,000 persons exposed or that have exposure concentrations greater than reference*

*standards, examine and choose risk reduction options in light of total facility risks and public health context.*

In accordance with CAA section 112(f)(2), EPA will consider the estimated cancer risks for facilities and implement management options that ensure an “ample margin of safety” as defined in the 1989 benzene NESHAP. The two-step benzene approach, described in detail in Section 2.1, is generally consistent with the Commission’s recommendation, although it does not incorporate a “flexible bright line” of  $10^{-5}$  (CRARM 1997b, p. 110). As discussed in Section 4.1.1, the Agency is developing risk management frameworks for non-cancer effects and carcinogens analyzed by an MOE approach.

EPA may consider total facility risks and public health context in risk management decisions when doing so will ensure that the concept of ample margin of safety is maintained.

- *Consider reduction of residual risks from source categories of lesser priority.*

EPA interprets this statement to say that the Agency should address highest risk source categories first.

The Agency will prioritize source categories for evaluation under the residual risk program to the extent possible, given data limitations and legislative time constraints. The goal of prioritizing will be to address source categories with higher risk first. EPA will use information from the Agency’s developing air toxics strategy and data gathered in the problem formulation part of the risk assessments (Exhibit 16) to help prioritize source categories.

#### **5.4 Residual Ecological Risk Decision Framework**

As discussed in Section 3.2, the Agency’s ecological risk assessment framework (EPA 1992a) and proposed guidelines (EPA 1996c) provide a broad perspective on the development and conduct of ecological risk assessments, as well as substantial detail on the key elements and issues required under each phase of an assessment. The iterative decision framework for evaluating residual ecological risks from HAP releases to the environment is depicted along-side the framework for evaluation of residual human health risks in Exhibit 16. In this section, we expand on the analyses applicable to the residual ecological risk assessment, including: (1) problem formulation, (2) analysis of ecological exposure and effects, and (3) risk characterization. Within the description of each phase, we indicate the considerations relevant to screening-level analyses (first tier) and those more applicable to refined analyses (subsequent tiers). We conclude this section with discussions of the approach currently under consideration by EPA (Section 5.4.4) and the decisions to be made on the basis of the risk characterization, as depicted in the diamonds in Exhibit 16 (Section 5.4.5).

#### 5.4.1 Problem Formulation

Problem formulation sets the context and scope of the ecological risk assessment. It includes selecting assessment endpoints and developing a conceptual model. At the screening level, it also includes an evaluation of the potential for specific HAPs to accumulate in the environment, which influences the need for multimedia analyses, and the relative toxicity of HAPs that partition into the same environmental media.

#### Screening Chemicals for Potential Accumulation in the Environment

To identify HAPs that are likely to accumulate in the environment, and thus potentially pose risks (ecological and/or human health) via food chains and other environmental media, the most important HAP characteristics are environmental persistence and bioaccumulation potential:

<b>environmental persistence</b>	If field data, chemical property data, or inference from chemical structure suggest that the HAP will persist in the environment for several weeks to several years (or longer), then a multimedia analysis might be necessary. For persistent and non-volatile HAPs, it is likely that the HAP will be deposited and accumulate over time in aquatic and terrestrial systems downwind of the source.
<b>bioaccumulation</b>	If field data, laboratory data, models (regression or food web), and/or the log $K_{ow}$ suggest that the HAP might accumulate in plant or animal tissues, then a food chain analysis might also be needed. Various cutoff values for screening bioaccumulation potential have been used. For example, the <i>Final Water Quality Guidance for the Great Lakes System</i> (EPA 1995h) used a bioaccumulation factor (BAF) in fish of 1,000 to identify bioaccumulative chemicals and log $K_{ow}$ values from 3.0 to 5.0 have been used to identify constituents likely to bioaccumulate in aquatic and terrestrial ecosystems (e.g., Connell 1988; Garten and Trabalka 1983; Suter 1993).

Where possible in the screening assessment, environmental characteristics that influence the behavior of a HAP in different media (e.g., persistence in water versus air) should be identified. For example, if a HAP is readily degraded by hydrolysis in surface water, aquatic life might not be at risk even if the HAP is toxic and persistent in air and deposits to surface waters, into which it readily partitions. In the more refined tiers of the ecological risk assessment, a literature search and review of studies that describe ecological impacts that have been clearly attributed to the HAP, or field measurement studies that indicate environmental “sinks” for the pollutant (i.e., in what environmental compartment(s) is the pollutant likely to accumulate), can be useful.

#### Screening Chemicals for Relative Toxicity

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For some source categories, several HAPs might be released. It is possible that the environmental behavior of several HAPs is such that they are expected to partition into the same environmental medium. If information is available to indicate that one or a few of those HAPs are much more toxic to ecological communities in contact with that medium than the remaining HAPs, then it might be possible to focus the screening assessment on the most toxic of those HAPs. If, in the screening analysis, the most toxic of those HAPs indicate no risks, then the less toxic HAPs would not need to be evaluated further.

### **Selecting Assessment Endpoints**

As described in Section 5.3, the overall risk management goal for the residual risk assessment is “to prevent an adverse environmental effect.” That goal can be rephrased as “to prevent adverse effects in ecological receptors from exposure to HAP releases.” Thus, the broad management goal encompasses potentially any type of ecosystem and the components of that ecosystem needed to maintain it. A main purpose of the initial tier (screening-level) of the ecological risk assessment is to screen out those HAPs and sources of HAPs that are unlikely to pose threats to ecological receptors based on readily available information. Because information on the habitats and ecosystems surrounding individual facilities of a source category generally is not readily available, for purposes of the screen, EPA assumes the presence of generic ecological receptors.

EPA assumes, for purposes of screening, that if the most sensitive species known to occur within an ecological community is protected from adverse effects caused by a HAP, the structure, and therefore the function, of the community also will be protected. Protection of the ecosystem as a whole is inferred from the protection of its component communities. These assumptions are consistent with those made by the Office of Water in developing ambient water quality criteria for the protection of aquatic life and with those made by the Office of Solid Waste in developing a variety of screening ecotoxicity criteria.

Information on the potential for HAPs to accumulate in the environment can be used to narrow a comprehensive set of assessment endpoints for the ecological risk screen. Given that HAPs are initially released to the air, the most important question for problem formulation at the screening level is the degree to which the HAPs might persist and partition into other environmental media. If a HAP is unlikely to accumulate in environment, then only those ecological communities that come into direct contact with HAPs in the air need be considered. The question of whether a multipathway analysis is needed is also asked during problem formulation in the screening-level human health risk assessment.

### **Developing the Conceptual Model**

As described in Sections 3.2.1, the conceptual model for a residual ecological risk assessment includes a description of the sources of HAP releases, information on emission rates, and a description of exposure pathways, assessment endpoints, and the measures that will be used

to evaluate the assessment endpoints. Multimedia analyses are likely to be needed for many of the persistent HAPs, whereas only air might need to be considered for some short-lived HAPs. For those HAPs that are not expected to accumulate in the environment, either locally or regionally, the conceptual model is relatively simple, and can be assumed to involve direct exposure of plant foliage to the air and inhalation of air by terrestrial animals. For those HAPs that might accumulate in other environmental media (e.g., in water, sediments, soil, or plants), a multimedia exposure model with the appropriate receptor communities will be needed. For HAPs that are likely to partition into sediments and soils, receptors of concern include the benthic aquatic community, the soil macro- and microinvertebrate community, and plants. For HAPs that are likely to partition into water, the benthic and free-swimming aquatic communities should be included. For HAPs that might bioconcentrate or bioaccumulate in aquatic organisms, the animals that feed on those organisms should be considered (e.g., piscivorous wildlife). For HAPs that might bioaccumulate in terrestrial plants, herbivorous animals should be included in the conceptual model.

#### **5.4.2 Analysis Phase**

The analysis phase of the ecological risk assessment involves two main steps: estimating HAP concentrations in the environment (including biota, where appropriate) and developing ecotoxicity benchmarks and exposure-response profiles. In the initial screening assessment, EPA generally intends to use point estimates for both the HAP concentrations in the environment and for ecological effects. The point estimates of ecological effects are referred to as ecotoxicity benchmarks. For the more refined assessments, spatial and temporal patterns of HAP contamination of the environment and more complete exposure-response profiles will be considered.

##### **Estimating HAP Concentrations in the Environment**

Based on the conceptual model developed during problem formulation, a single or multimedia model will be required to estimate HAP concentrations in environmental media of concern (i.e., air only, or air and soils, sediments, surface waters, and biota). Basically, the same considerations apply to the screening exposure analyses for the human health risk and ecological risk assessments. For multimedia analyses, simple conservative bioaccumulation factors and models of transfer of HAPs from air and soils to plants should suffice. In a more refined assessment, more sophisticated models can be used to simulate the fate and transport of contaminants in the ecosystem of concern, or concentrations in environmental media might actually be measured in the field and mapped to depict the contamination pattern at the specific site.

### **Developing Ecotoxicity Benchmarks and Stressor-Response Profiles**

In the screening-level assessment, an ecotoxicity benchmark is needed for each combination of environmental medium and ecological community described by the generic assessment endpoints in the conceptual model. For a persistent HAP that might partition into all environmental media, screening ecotoxicity benchmarks could be needed for all of the following media/receptor combinations:

- air/terrestrial animals exposed via inhalation;
- air/plants with their foliage exposed to the air;
- water/aquatic biota exposed via direct contact with water;
- sediments/benthic aquatic biota exposed via direct contact with sediments;
- soil/soil macro- and micro-invertebrates; and
- soil/plants.

For each medium/receptor combination identified above, the screening-level ecotoxicity benchmarks are expressed as a concentration of the HAP in the environmental medium. As noted in Section 5.4.1, the benchmarks are intended to be protective of a most sensitive species in any of the generic communities identified above.

For a persistent HAP that might also bioaccumulate in plants or animals, a reference dose considered protective of wildlife that feed on those plants or animals would be needed along with information on food ingestion rates for sensitive and most exposed animal species and information on the degree of bioaccumulation in appropriate trophic components. Examples of that approach for aquatic systems can be found in the Great Lakes Water Quality Initiative for mercury, DDT, PCBs, and 2,3,7,8-TCDD (EPA 1995d,h) and for terrestrial systems in the EPA methods of assessing exposures to combustor emissions (EPA 1993b).

The first step in obtaining the screening ecotoxicity benchmarks identified above is to determine if any existing ecotoxicity benchmarks are appropriate to the current purpose. Several efforts to develop such benchmarks currently are ongoing. Any existing ecotoxicity benchmarks should be evaluated to determine their applicability to a screening or to a more refined analysis. The screening analyses should use conservative benchmarks derived from no-observed-effect levels (NOELs) for a most sensitive species for the community in question. Use of benchmarks derived from those data might overestimate risk, but should seldom underestimate it. Other options are available for the more refined analyses, taking into account costs, safety, energy, and other relevant factors, as required by law.

If appropriate screening-level ecotoxicity benchmarks are not available for a specific HAP, risk assessors can develop benchmarks from toxicity studies (see types described in Section 3.3). The most appropriate laboratory tests for screening-level analyses are those that measure effects on survival, growth, and reproduction. The order of preference for the test results is NOEL, lowest-observed-effect-level (LOEL), and estimated low effect levels (e.g., LC<sub>LO</sub>). Uncertainty

factors can be used to estimate a NOEL from higher effect levels or to estimate effects possible from chronic exposure from tests using shorter exposure durations. In addition, uncertainty factors can be used to estimate a NOEL for a most sensitive species from data on only a few species in the community in question.

In the more refined analyses, benchmarks might be calculated for site-specific ecological receptors depending on the importance of those receptors to the local ecosystem, or a benchmark might be calculated for an endpoint not previously evaluated. For example, while endpoints used to develop screening-level benchmarks are based on survival, growth, and reproductive success, a benchmark for a threatened or endangered species, a valuable game species (e.g., trout), or an ecologically key species (e.g., wolf) might be based on an endpoint that is relevant to individual organism health (e.g., a neurological deficit) rather than to population maintenance. On the other hand, benchmarks based on higher effect levels (e.g., 20 to 50 percent or higher) might be appropriate for species for which great functional redundancy exists in the ecosystem (e.g., different herbaceous plants; see Lawton and Brown, 1994).

In the more refined analyses, development of stressor-response curves, instead of point estimates of effect, can provide more information for and flexibility in evaluating risks. For example, stressor-response curves can allow a description of the areal extent of a community that might be affected to differing degrees (e.g., 40 percent mortality of soil invertebrates over 10 acres, 20 percent mortality over the surrounding 100 acres, and less than 10 percent mortality of soil invertebrates in areas beyond those 110 acres).

The more refined analyses also will need professional judgment as to what effect levels from experimental studies are suggestive of adverse ecological effects at a particular site (i.e., what effects are “significant”). Natural populations and communities usually can compensate for some degree of loss in survivorship or reproduction. The ability for populations to compensate for some loss depends on species’ characteristics (e.g., longevity, growth rate, reproductive rate) and characteristics of the ecosystem and communities in which the species exists (e.g., food abundance, presence of competitors, natural stress levels). Plants tend to be very resilient and able to tolerate or compensate for a wide range of natural (e.g., drought) and anthropogenic stressors. All “natural” populations and communities undergo changes on at least a seasonal basis, and ecosystems can exist in many different states, all of which might be “healthy” and likely to persist over time. Currently, there is little guidance and a lack of consensus among the scientific community as to what constitutes ecological significance.

### **5.4.3 Risk Characterization**

The results of the exposure and ecological effects assessments are integrated to characterize risk. In the screening-level ecological risk characterization, the maximum HAP concentrations estimated for the various environmental media are compared to the appropriate screening-level ecotoxicity benchmarks for each ecological community specified in the conceptual model. The ratio of the estimated environmental concentration to the ecotoxicity benchmark is called the hazard quotient. A risk is assumed when the hazard quotient exceeds 1. If multiple HAPs can affect the same receptors, the hazard index (HI) approach should be used. The HI equals the sum of the hazard quotients for individual HAPs that apply to the same receptors (see Section 3.4.3).

If a more refined analysis is needed, more realistic (i.e., less generic) approaches can be used to characterize risks. For example, one can compare an ecotoxicity benchmark to an average instead of maximum estimated HAP concentration, using an ecologically relevant area over which to average the concentrations. One can compare a series of isopleths (i.e., lines of constant concentration) of estimated HAP concentrations in the environment to stressor-response curves. For the more refined analyses of specific sites, mapping the overlap of isopleths of estimated or measured HAP concentrations with the location of ecological receptors can be helpful in evaluating the significance of the risks. For example, population-level models might be adapted for an ecorisk application to delineate the impact of a chemical stressor on population dynamics over space and time. Such tools have already been used successfully in ecological risk assessments, particularly for fish populations (see Suter 1993). Information to be included in such refined risk characterizations would also include the local, State, Tribal, and/or regional ecological value or significance of the ecological entities at risk.

Without calibrated or validated population models, professional judgment is needed to estimate the ecological significance of contaminant concentrations that exceed levels associated with varying magnitudes of effect on different species or communities. Unless an endangered or threatened species is at issue, society is generally not concerned with the death of individual animals. For other species, it is unlikely that a few percent additional mortality of individuals could result in population-level effects that might impair ecosystem structure and function. However, it is extremely difficult to estimate how much additional contaminant-induced mortality or reduced reproductive success a population can

#### **EXAMPLES OF CONSIDERATIONS FOR DETERMINING ECOLOGICAL SIGNIFICANCE**

- ▶ What is the areal extent of the benchmark exceedance?
- ▶ What proportion of the habitat is affected at local, county, and State levels?
- ▶ Are the exposure concentrations and benchmarks above background levels for the area of interest?
- ▶ What types of ecological impacts have been associated with this pollutant or similar pollutants in the past?
- ▶ Is the benchmark or stressor-response curve based on high quality data (i.e., is there a high degree of confidence in the benchmark)?
- ▶ What are the costs, energy, safety, and other relevant considerations required for decision-making?

compensate for before population levels begin to decline, particularly if the population is subject to other stresses.

Nonetheless, in the more refined ecological risk assessment(s), several considerations are helpful in estimating ecological significance, as shown in the accompanying text box. Professional judgment is required in weighing such considerations to develop conclusions concerning the significance of the ecological risks. Stakeholder input also can be valuable in characterizing the societal importance of the ecosystems at risk.

**Exhibit 18** summarizes the assumptions and criteria used to evaluate residual ecological risks for the screening analysis and for the more refined analyses. The exhibit is organized according to the phases of the ecological risk assessment process as described in Section 3.2.

#### **5.4.4 Approach Currently Under Consideration**

EPA currently is testing a method for determining environmental residual risks. As a first step, a risk assessment decision framework specifically targeted toward HAPs was developed in-house. The framework presented a tiered approach to evaluating residual risks that facilitated different levels of assessment (e.g., preliminary screening to site-specific), as needed. This framework has been independently reviewed and determined to conform with the EPA guidelines (EPA 1996c).

Tier One of the framework provides for a screen of HAPs based on their environmental behavior. It undertakes to determine if the potential exists for adverse effects due to a particular HAP's ability to persist, bioaccumulate, or exhibit acute toxicity. Those HAPs that fail the screen (i.e., persist, bioaccumulate, and/or exhibit toxicity) undergo closer scrutiny in a second tier. This second tier is a more intensive screening step that employs multipathway analysis to estimate if, and to what extent, generic ecological receptors may be exposed to HAPs.

In Tier Two, the approximate physical locations of the HAP emission sources are determined from available information such as emissions profiles derived from the development of MACT source categories, the Background Information Documents (BID) for proposed MACT standards, and MACT Model Plants data. Using the approximate sites, a generic ecosystem model including representative environmental and ecological receptors for the sites at risk is developed. Also, the appropriate benchmarks for both environmental media and ecological receptors that describe contaminant levels for the selected HAPs presumed to represent a no-adverse-effects threshold are identified. The exposure and potential impact is then modeled and compared to predetermined assessment endpoints. The third tier of the proposed approach is recommended for those HAPs that surpass a particular assessment endpoint and are determined

**EXHIBIT 18**  
**SUMMARY OF ASSUMPTIONS AND CRITERIA FOR EVALUATING ECOLOGICAL RISKS**

<b>Component of the Risk Assessment</b>	<b>Screening Level</b>	<b>Refined<sup>1</sup></b>
Problem Formulation	<ul style="list-style-type: none"> <li>▶ Based on generic aquatic and terrestrial ecosystems assumed to be near all source category facilities</li> <li>▶ HAPs screened for those that might require multipathway analyses</li> <li>▶ Generic multimedia conceptual model simplified based on HAP characteristics and likely exposure pathways</li> <li>▶ Generic assessment endpoints of maintaining ecological community structure and function are used for the communities that might be exposed</li> </ul>	<ul style="list-style-type: none"> <li>▶ Based on more site-specific information on ecosystems, habitats, and species near the facilities of concern</li> <li>▶ Results of screening analysis used to identify HAPs and exposure pathways of concern</li> <li>▶ More site-specific conceptual model developed based on results of screening analysis and site-specific information</li> <li>▶ Correspondingly more refined assessment endpoints are developed</li> </ul>
Analysis Phase: Exposure Assessment	<ul style="list-style-type: none"> <li>▶ Simple conservative assumptions and screening-level exposure models are used</li> <li>▶ Conservative values from the literature are assumed for factors such as bioavailability and bioaccumulation</li> <li>▶ Locations with maximum estimated HAP concentration is used to estimate exposure</li> </ul>	<ul style="list-style-type: none"> <li>▶ More refined assumptions, site-specific data, and refined exposure models are used</li> <li>▶ More representative values from the literature or actual measurements from the field are used for factors such as bioavailability and bioaccumulation</li> <li>▶ Spatial and temporal extent and magnitude of contamination are estimated</li> </ul>
Analysis Phase: Ecological Effects Assessment	<ul style="list-style-type: none"> <li>▶ Screening-level ecotoxicity benchmarks are identified or developed as point estimates of no-observed-effect levels for the most sensitive species in the generic communities</li> </ul>	<ul style="list-style-type: none"> <li>▶ Refined ecotoxicity benchmarks are identified or developed as point estimates of low-observed-effect-levels for the assessment endpoints identified under problem formulation</li> <li>▶ As data permit, full stressor-response curves might be developed</li> <li>▶ Actual field evaluation of ecological condition near some facilities might be performed</li> </ul>
Risk Characterization	<ul style="list-style-type: none"> <li>▶ Additivity of all HAPs is assumed</li> <li>▶ <math>HI &lt; 1</math> acceptable; <math>\geq 1</math> leads to a reexamination of conservative assumptions and, if the HI continues to exceed 1, to a more refined analyses</li> <li>▶ Potential ecological significance of effects is not evaluated</li> </ul>	<ul style="list-style-type: none"> <li>▶ More detailed treatment of mixtures</li> <li>▶ <math>HI &lt; 1</math> acceptable; <math>\geq 1</math> might be acceptable depending on ecological significance</li> <li>▶ Potential ecological significance of effects is evaluated based on a number of factors, including areal extent and magnitude of estimated effects on assessment endpoints and local, State, Tribal, or regional significance of the assessment endpoints</li> </ul>

<sup>1</sup> Refined assessment based on more detailed and site-specific, and less conservative, estimated HAP exposures and presence of ecological receptors throughout the spatial area of impact.

to pose significant concern to an ecosystem. This tier consists of a site-specific multipathway risk assessment, or similar analysis, which results in a detailed determination of residual risks. The results of this determination coupled with other factors, such as costs, safety, and energy will then be used to guide EPA's decision to protect against an adverse environmental effect.

#### **5.4.5 Decisions Based on Risk Characterization**

At the end of the screening-level risk characterization, if none of the estimated environmental concentrations exceeds the corresponding benchmarks, the conservative risk screen indicates that the source category does not pose a risk of "an adverse environmental effect." The results of the screening analysis should be documented, and the ecological risk assessment process would stop. On the other hand, there might be one or more HAPs and combinations of exposure media and ecological communities for which the exposure concentration exceeds the screening ecotoxicity benchmark (i.e., the hazard quotient is greater than 1) or for which the sum of the hazard quotients that apply to the same communities exceeds 1. If any sources or HAPs result in exposures in excess of the appropriate ecotoxicity screening benchmark, further analysis is warranted.

If the exceedance of a screening-level ecotoxicity benchmark is small (e.g., less than an order of magnitude), it is worth reexamining all of the conservative assumptions used in the screening analyses to see if a more realistic combination of fate and transport parameters or more realistic values for other key parameters would eliminate the exceedance. Common conservative assumptions that should be reexamined at this point include, among others, use of conservative bioaccumulation factors from the literature, assuming that bioavailability is 100 percent, or assuming that 100 percent of a metal is present in its most toxic form (e.g., methyl mercury instead of elemental mercury).

If the exceedance of a screening-level ecotoxicity benchmark is large (e.g., more than an order of magnitude) or remains after selected less conservative assumptions are used, then a more refined risk assessment is indicated. If only one or a few of the facilities within a source category are likely to cause the exceedance, then a more refined assessment for those individual facilities using site-specific information might be appropriate. If several facilities are likely to be at issue, a more refined analysis across the board might be needed. At this stage, it can be particularly helpful to involve stakeholders, letting them review the results of the screening risk analysis and asking them which risk characterization and risk management questions they would like to see answered.

The decision on how to exit the diamond in Exhibit 16 depends on the level of confidence that is required to make the decision of "no further action" and on the level of accuracy of the risk predictions that is needed to evaluate options for reducing risks. In addition, how much the risk estimates can be improved by refining the analysis is an important consideration.

Evaluating the sensitivity of the risk results to different components of the risk analysis can help identify which components are most important and allow the assessors to refine the more sensitive analyses or assumptions sequentially. If it appears that some site-specific information will need to be collected in the field (e.g., identify and evaluate the ecosystems surrounding a facility and the pattern of contamination around the facility), the problem formulation step and conceptual model will need to be refined as thoroughly as possible, and an analysis plan should be developed for the field data collection and assessment. During this problem formulation, assessment endpoints will be defined on a site-specific basis. It might be possible to identify species that require a higher level of protection (e.g., game fish) than species for which greater functional redundancy exists (e.g., forage fish, for which many species can play a similar functional role in the ecosystem). Moreover, on a site-specific basis, endpoints other than direct toxicological effects might be considered, such as a change in algal species composition in response to a chemical stressor that results in a decline in water quality.

As described in Section 5.3, the ecological risk assessment results are one of several inputs to the final risk management decision. Other statutory considerations that are considered include cost, energy, safety, and other relevant factors.

## **5.5 Summary of Residual Risk Strategy**

Following the framework provided by Congress in CAA section 112(f), EPA has developed a strategy to identify, assess, and manage the residual risks associated with air toxics emissions following the application of MACT standards to source categories. The strategy is guided by sections 112(f)(2) through (6), as well as influenced by the recent recommendations made by the NRC (NRC 1994) and the Risk Commission (CRARM 1997a,b), and it incorporates EPA's current risk assessment and risk management policies, published guidelines, and methods. In short, the strategy consists of a tiered, iterative assessment of the human health and environmental risks resulting from both direct and multipathway exposures to HAPs following MACT implementation, leading ultimately to decisions on whether additional emission reductions are needed for individual source categories. Key steps in the strategy include problem formulation, data collection, risk analysis, and risk management/risk reduction decision-making. The human health risk management decision criteria are based on the "ample margin of safety" principles, first laid out in EPA's 1989 national emission standard for benzene and affirmed in the 1990 CAA Amendments, and the environmental decision criteria are based on the "...prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect" language in the statute. The residual risk strategy is intended to provide EPA appropriate flexibility in its decisions while ensuring that the public and environmental health is protected from air toxics as envisioned by Congress in the CAA.

## References

Agency for Toxic Substances and Disease Registry (ATSDR). 1993a. Toxicological profile for cadmium. Atlanta: U.S. Department of Health and Human Services, Public Health Service.

Agency for Toxic Substances and Disease Registry (ATSDR). 1993b. Toxicological profile for lead. Atlanta: U.S. Department of Health and Human Services, Public Health Service.

Agency for Toxic Substances and Disease Registry (ATSDR). 1994. Toxicological profile for mercury. Atlanta: U.S. Department of Health and Human Services, Public Health Service.

Baird, S.J., J.T. Cohen, J.D. Graham, A.I. Shlyakhter, and J.S. Evans. 1996. Non-cancer risk assessment: A probabilistic alternative to current practice. *Human and Ecological Risk Assessment* 2(1):79-102.

Binkova, B., J. Lewtas, I. Miskvova, J. Lenicek, and R. Sram. 1995. DNA adducts and personal air monitoring of carcinogenic polycyclic aromatic hydrocarbons in an environmentally exposed population. *Carcinogenesis (England)* 16(5):1037-46.

Brown, L.M., L.M. Pottern, and W.J. Blot. 1984. Lung cancer in relation to environmental pollutants emitted from industrial sources. *Environ. Res.* 34:250-261.

Bustueva, K.A., B.A. Revich, and L.E. Bezpalko. 1994. Cadmium in the environment of three Russian cities and in human hair and urine. *Arch. Environ. Health* 49(4):284-288.

Commission on Risk Assessment and Risk Management (CRARM). 1996. Risk assessment and risk management in regulatory decision-making. Draft report. Washington, DC.

Commission on Risk Assessment and Risk Management (CRARM). 1997a. Framework for environmental health risk management. Final report, Volume 1.

Commission on Risk Assessment and Risk Management (CRARM). 1997b. Risk assessment and risk management in regulatory decision-making. Final report, Volume 2.

Connell, D.W. 1988. Bioaccumulation behavior of persistent organic chemicals with aquatic organisms. *Rev. of Environ. Contamin. and Toxicol.* 101:118-154.

Edmonds, L.D., C.E. Anderson, J.W. Flynt Jr., and L.M. James. 1978. Congenital central nervous system malformations and vinyl chloride monomer exposure: A community study. *Teratology* 17:137-142.

Finley, B. and D. Paustenbach. 1994. The benefits of probabilistic exposure assessment: Three case studies involving contaminated air, water, and soil. *Risk Analysis* 14(1):53-73.

Frost, F., L. Harter, S. Milham, R. Royce, A. Smith, J. Hartley, and P. Enterline. 1987. Lung cancer among women residing close to an arsenic emitting copper smelter. *Arch. Environ. Health* 42:148-152.

Garten, C.T., Jr. and J.R. Trabalka. 1983. Evaluation of models for predicting terrestrial food chain behavior of xenobiotics. *Environ. Sci. Technol.* 17(10):590-595.

Hughes, J.P., L. Polissar, and G. Van Belle. 1988. Evaluation and synthesis of health effects studies of communities surrounding arsenic producing industries. *Int. J. Epidemiol.* 17:407-413.

Lawton, J.H., and V.K. Brown. 1994. Redundancy in ecosystems. pp. 255-270. In: *Biodiversity and ecosystem function*, E.D. Schulze and H. A. Mooney, eds. Springer-Verlag, Berlin Heidelberg, Germany.

Morgan, G. and M. Henrion. 1990. *Uncertainty: A guide to dealing with uncertainty in quantitative risk and policy analysis*. Cambridge University Press.

National Research Council (NRC). 1983. *Risk assessment in the federal government: Managing the process*. National Academy Press, Washington, DC.

National Research Council (NRC). 1993b. *Guidelines for developing community emergency exposure levels for hazardous substances*. National Academy Press, Washington, DC.

National Research Council (NRC). 1994. *Science and judgment in risk assessment*. National Academy Press, Washington, DC.

National Research Council (NRC). 1996. *Understanding risk*. National Academy Press, Washington, DC.

Natural Resources Defense Council (NRDC), Inc. v. EPA. 1987. 824 F.2d at 1146.

Nordstrom, S., L. Beckman, and I. Nordenson. 1978. Occupational and environmental risks in and around a smelter in northern Sweden. I. Variations in birthweight. *Hereditas* 88:43-46.

Pershagen, G. 1985. Lung cancer mortality among men living near an arsenic emitting smelter. *Am. J. Epidemiol.* 122:684-694.

Rosenman, K.D., J.E. Rizzo, M.G. Conomos, and G.J. Halpin. 1989. Central nervous system malformations in relation to polyvinyl chloride production facilities. *Arch. Environ. Health* 44(5):279-282.

Shy, C.M. 1993. Epidemiological studies of neurotoxic, reproductive, and carcinogenic effects of complex mixtures. *Environ Health Perspect* 101(Suppl. 4):183-186.

- STAPPA/ALAPCO. 1989. Toxic air pollutants: State and local regulatory strategies.
- Suter, G.W., II. 1993. Ecological risk assessment. Lewis Publishers, Chelsea, MI.
- Theriault, G., H. Iturra, and S. Gingras. 1983. Evaluation of the association between birth defects and exposure to ambient vinyl chloride. *Teratology* 27:359-370.
- U.S. EPA. 1984b. Risk Assessment and Management: Framework for Decision Making. Office of Policy, Planning, and Evaluation, Washington, DC. EPA-600/9-85-002.
- U.S. EPA. 1986a. Guidelines for exposure assessment. *Federal Register* 51:34042-34054. September 24, 1986.
- U.S. EPA. 1986b. Guidelines for mutagenicity risk assessment. *Federal Register* 51:34006-34012. September 24, 1986.
- U.S. EPA. 1986c. Guidelines for carcinogen risk assessment. *Federal Register* 51:33992-34003. September 24, 1986.
- U.S. EPA. 1986d. Guidelines for the health risk assessment of chemical mixtures. *Federal Register* 51:34014-34025. September 24, 1986.
- U.S. EPA. 1986e. Users manual for the Human Exposure Model (HEM). OAQPS, Research Triangle Park, NC. EPA-540/5-86-001. June 1986.
- U.S. EPA. 1986f. Guidelines for deriving numerical water quality criteria for the protection of aquatic organisms and their uses. Office of Water Regulations and Standards, Washington, DC.
- U.S. EPA. 1986g. Standard evaluation procedure for ecological risk assessment. Office of Prevention, Pesticides, and Toxic Substances and Office of Pesticide Programs, Washington, DC. EPA-540/9-85-001.
- U.S. EPA. 1988a. National emission standards for hazardous air pollutants. *Federal Register* 53(145):28496, Proposed Rule and Notice of Public Hearing. July 28, 1988.
- U.S. EPA. 1988b. Proposed guidelines for exposure-related measurements. *Federal Register* 53:48830-48853. December 2, 1988.
- U.S. EPA. 1989b. National Emission Standards for Hazardous Air Pollutants; Benzene. *Federal Register* 54(177):38044-38072, Rule and Proposed Rule. September 14, 1989.
- U.S. EPA. 1989c. Risk assessment guidance for Superfund, Volume I: Human health evaluation manual. Office of Emergency and Remedial Response.
-

U.S. EPA. 1989d. Risk assessment guidance for Superfund, Volume II: Environmental evaluation manual. Office of Emergency and Remedial Response.

U.S. EPA. 1990a. Methodology for assessing health risks associated with indirect exposure to combustor emissions. Office of Research and Development and Office of Health and Environmental Assessment, Washington, D.C. EPA-600/6-90-003.

U.S. EPA. 1991a. Guidelines for developmental toxicity risk assessment. *Federal Register* 56:63798-63826.

U.S. EPA. 1992a. Framework for ecological risk assessment. Risk Assessment Forum, Office of Research and Development, Washington, D.C. EPA-630/R-92-001. February 1992.

U.S. EPA. 1992b. A tiered modeling approach for assessing the risks due to sources of hazardous air pollutants. Office of Air Quality Planning and Standards, RTP, NC. EPA-450/4-9-001.

U.S. EPA. 1992c. Guidelines for exposure assessment. *Federal Register* 57:22888-22938. May 29, 1992.

U.S. EPA. 1992d. Guidance on risk characterization for risk managers and risk assessors (“Habicht Memorandum”). Risk Assessment Council, Washington, DC. February 26, 1992.

U.S. EPA. 1993a. A descriptive guide to risk assessment methodologies for toxic air pollutants. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-453/R-93-038.

U.S. EPA. 1993b. Addendum to the methodology for assessing health risks associated with indirect exposure to combustor emissions. Office of Research and Development, Washington, D.C. EPA-600/AP-93-003.

U.S. EPA. 1993d. A review of ecological assessment case studies from a risk assessment perspective. Risk Assessment Forum, Office of Research and Development, Washington, DC. EPA-630/R-92-005. May 1993.

U.S. EPA. 1993e. National emission standards for hazardous air pollutants schedule for the promulgation of emission standards under section 112(e) of the Clean Air Act Amendments of 1990. *Federal Register* 58:63941-63954. December 3, 1993.

U.S. EPA. 1994a. Exposure assessment guidance for RCRA hazardous waste combustion facilities. Office of Solid Waste and Emergency Response, Washington, D.C. EPA-530/R-94-021.

U.S. EPA. 1994b. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Washington, DC.

U.S. EPA. 1994c. Methods for exposure-response analysis and health assessment for acute inhalation exposure to chemicals: Development of the acute reference exposure. Draft Working Paper. Office of Health and Environmental Assessment, Research Triangle Park, NC.

U.S. EPA. 1994g. A review of ecological assessment case studies from a risk assessment perspective, Volume II. Risk Assessment Forum, Office of Research and Development, Washington, DC. EPA-630/R-94-003. July 1994.

U.S. EPA. 1994h. National Human Activity Pattern Survey (NHAPS) – Use of nationwide activity data for human exposure assessment (W.C. Nelson, Project Officer, EPA/ORD, RTP, NC.). Presented at American Waste Management Association meeting. Cincinnati, OH. June. Paper #94-WA75A.01

U.S. EPA. 1994i. Estimating exposure to dioxin-like compounds (three volumes). External review draft. EPA-600/6-88-005Ca.

U.S. EPA. 1995a. Guidance for risk characterization (“Browner Memorandum”). Science Policy Council, Washington, DC.

U.S. EPA. 1995b. Proposed guidelines for neurotoxicity risk assessment. *Federal Register* 60(192):52032-52056. Office of Research and Development, Research Triangle Park, NC.

U.S. EPA. 1995c. Technical support document for the Hazardous Waste Identification Rule: Risk assessment for human and ecological receptors. Office of Solid Waste. August 1995.

U.S. EPA. 1995d. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,6,8-TCDD, PCBs. Office of Water. EPA-820/B-95-008.

U.S. EPA. 1995e. Summary and analysis of available air toxics health effects data. Final draft. Prepared by ICF Incorporated for OAQPS, U.S. EPA. September 1995.

U.S. EPA. 1995h. Final water quality guidance for the Great Lakes System. *Federal Register* 60:15366, Final Rule. March 23, 1995.

U.S. EPA. 1996b. Proposed guidelines for carcinogen risk assessment. Office of Research and Development, Washington, DC. EPA-600/P-92-003C.

U.S. EPA. 1996c. Proposed guidelines for ecological risk assessment. *Federal Register* 61:47552. September 9, 1996. Risk Assessment Forum, Office of Research and Development, Washington, DC. EPA-630/R-95-002B. August 1996.

U.S. EPA. 1996d. Proposed guidelines for reproductive toxicity risk assessment. EPA-630/R-96-009. September 1996.

U.S. EPA. 1997a. Guiding principles for Monte Carlo analysis. EPA-630/R-97-001.

U.S. EPA. 1997b. Policy for use of probabilistic analysis in risk assessment. May 15, 1997.

U.S. EPA. 1997c. Protocol for screening-level human health risk assessment at hazardous waste combustion facilities. Internal Review Draft. Office of Solid Waste. February 28, 1997.

U.S. EPA. 1997d. Exposure factors handbook. Office of Research and Development, Washington, DC.

U.S. EPA. 1997e. Guidance on cumulative risk assessment, Part 1, Planning and scoping. Science Policy Council, Washington, DC.

U.S. EPA. 1997f. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. Environmental Response Team, Edison, NJ.

U.S. EPA. 1997g. Draft guidelines for risk assessment of chemical mixtures.

U.S. EPA. 1997h. National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances; Notice. *Federal Register* 62:58839-58851. October 30, 1997.

U.S. EPA. 1997i. Methodology for assessing health risks associated with multiple exposure to combustor emissions. Office of Research and Development. NCEA 0238 (Update to EPA/600/6-90/003). Cincinnati, OH.

U.S. EPA. 1997j. National air quality and emissions trends report, 1996. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-454/R-97-013. October 1997.

U.S. EPA. 1997k. Mercury Study Report to Congress. EPA-452/R-97-0003. December 1997.

U.S. EPA. 1997l. Deposition of Air Pollutants to the Great Waters. Second Report to Congress. EPA-453/R-97-011. June 1997.

U.S. EPA. 1998. Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units – Final Report to Congress. EPA-453/R-98-004. February 1998.

U.S. Senate. 1997. “A Bill to Provide for Analysis of Major Rules,” S. 981. Committee of Government Affairs. June 27, 1997.

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## **Appendix A**

### **Full Text of Clean Air Act Section 112(f)**

## **Appendix A**

### **Full Text of Clean Air Act Section 112(f)**

(f) Standard to Protect Health and the Environment. — (1) Report. — Not later than 6 years after the date of enactment of the Clean Air Act Amendments of 1990 the Administration shall investigate and report, after consultation with the Surgeon General and after opportunity for public comment, to Congress on —

(A) methods of calculating the risk to public health remaining, or likely to remain, from sources subject to regulation under this section after the application of standards under subsection (d);

(B) the public health significance of such estimated remaining risk and the technologically and commercially available methods and costs of reducing such risks;

(C) the actual health effects with respect to persons living in the vicinity of sources, any available epidemiological or other health studies, risks presented by background concentrations of hazardous air pollutants, any uncertainties in risk assessment methodology or other health assessment technique, and any negative health or environmental consequences to the community of efforts to reduce such risks; and

(D) recommendations as to legislation regarding such remaining risk.

(2) Emission Standards. — (A) If Congress does not act on any recommendation submitted under paragraph (1), the Administrator shall, within 8 years after promulgation of standards for each category or subcategory of sources pursuant to subsection (d), promulgate standards for such category or subcategory if promulgation of such standards is required in order to provide an ample margin of safety to protect public health in accordance with this section (as in effect before the date of enactment of the Clean Air Act Amendments of 1990) or to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect. Emission standards promulgated under this subsection shall provide an ample margin of safety to protect public health in accordance with this section (as in effect before the date of enactment of the Clean Air Act Amendments of 1990), unless the Administrator determines that a more stringent standard is necessary to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect. If standards promulgated pursuant to subsection (d) and applicable to a category or subcategory of sources emitting a pollutant (or pollutants) classified as a known, probable, or possible human carcinogen do not reduce lifetime excess cancer risks to the individual most exposed to emissions from a source in the category or subcategory to less than one in one million, the Administrator shall promulgate standards under this subsection for such source category.

(B) Nothing in subparagraph (A) or in any other provision of this section shall be construed as affecting, of applying to the Administrator's interpretation of this section, as in effect before the date of enactment of the Clean Air Act Amendments of 1990 and set forth in the Federal Register of September 14, 1989 (54 Federal Register 38044).

(C) The Administrator shall determine whether or not to promulgate such standards and, if the Administrator decides to promulgate such standards, shall promulgate the standards 8 years after promulgation of the standards under subsection (d) for each source category or subcategory concerned. In the case of categories or subcategories for which standards under subsection (d) are required to be promulgated within 2 years after the date of enactment of the Clean Air Act Amendments of 1990, the Administrator shall have 9 years after promulgation of the standards under subsection (d) to make the determination under the preceding sentence and, if required, to promulgate the standards under this paragraph.

(3) Effective date. — Any emission standard established pursuant to this subsection shall become effective upon promulgation.

(4) Prohibition. — No air pollutant to which a standard under this subsection applies may be emitted from any stationary source in violation of such standard, except that in the case of an existing source —

(A) such standard shall not apply until 90 days after its effective date, and

(B) the Administrator may grant a waiver permitting such source a period of up to 2 years after the effective date of a standard to comply with the standard if the Administrator finds that such period is necessary for the installation of controls and that steps will be taken during the period of the waiver to assure that the health of persons will be protected from imminent endangerment.

(5) Area sources. — The Administrator shall not be required to conduct any review under this subsection or promulgate emission limitations under this subsection for any category or subcategory of area sources that is listed pursuant to subsection (c)(3) and for which an emission standard is promulgated pursuant to subsection (d)(5).

(6) Unique Chemical Substances. — In establishing standards for the control of unique chemical substances of listed pollutants without CAS numbers under this subsection, the Administrator shall establish such standards with respect to the health and environmental effects of the substances actually emitted by sources and direct transformation byproducts of such emissions in the categories and subcategories.

## **Appendix B**

### **Preamble Excerpts from 1989 Benzene NESHAP**

**Appendix B**  
**Preamble Excerpts from 1989 Benzene NESHAP**

[Full text of Preamble Sections 1, 2, and 3 Only]

ENVIRONMENTAL PROTECTION AGENCY  
AGENCY: Environmental Protection Agency (EPA).

40 CFR Part 61  
National Emission Standards for Hazardous Air Pollutants;  
Benzene Emissions From Maleic Anhydride Plants,  
Ethylbenzene/Styrene Plants, Benzene Storage Vessels,  
Benzene Equipment Leaks, and Coke By-Product Recovery Plants

[AD-FRL-3620-4]  
RIN 2060-AC41

54 FR 38044

September 14, 1989

ACTION: Final rule.

**I. Summary of Decisions**

- Overview
- Background
- Selection of Approach
- Maleic Anhydride Process Vents
- Ethylbenzene/Styrene Process Vents
- Benzene Storage Vessels
- Coke By-Product Recovery Plants
- Benzene Equipment Leaks

**II. Background**

- Regulatory Background
- Public Participation
- Legal Framework Under Vinyl Chloride

**III. Application of Policy to Benzene Source Categories**

- Introduction
- Ethylbenzene/Styrene Process Vents
- Benzene Storage Vessels
- Coke By-Product Recovery Plants
- Benzene Equipment Leaks

**I. Summary of Decisions**

*Overview*

This section provides a description of the EPA's approach for the protection of public health under section 112. In protecting public health with an ample margin of safety under section 112, EPA strives to provide maximum feasible protection against risks to health from hazardous air pollutants by (1) protecting the greatest number of persons possible to an individual lifetime risk level no higher than approximately 1 in 1 million and (2) limiting to no higher than approximately 1 in 10 thousand the estimated risk that a person living near a plant would have if he or she were exposed to the maximum pollutant concentrations for 70 years. Implementation of these goals is by means of a two-step standard-setting approach, with an analytical first step to determine an "acceptable risk" that considers all health information, including risk estimation uncertainty, and includes a presumptive limit on maximum individual lifetime risk (MIR) of approximately 1 in 10 thousand. A second step follows in which the actual standard is set at a level that provides "an ample margin of safety" in consideration of all health information, including the number of persons at risk levels higher than approximately 1 in 1 million, as well as other relevant factors including costs and economic impacts, technological feasibility, and other

factors relevant to each particular decision. Applying this approach to the five benzene source categories in today's notice results in controls that protect over 99 percent of the persons within 50 kilometers (km) of these sources at risk levels no higher than approximately 1 in 1 million.

A principle that accompanies these numerical goals is that while the Agency can establish them as fixed numbers, the state of the art of risk assessment does not enable numerical risk estimates to be made with comparable confidence. Therefore, judgment must be used in deciding how numerical risk estimates are considered with respect to these goals. As discussed below, uncertainties arising from such factors as the lack of knowledge about the biology of cancer causation and gaps in data must be weighed along with other public health considerations. Many of the factors are not the same for different pollutants, or for different source categories.

### *Background*

On July 28, 1988, EPA proposed decisions on standards under Section 112 for five source categories of benzene. A principal aspect of the proposal, and the basis for the proposed decisions on the source categories, were four proposed approaches for decisions under Section 112 as mandated by the DC Circuit's decision in *NRDC v. EPA*, 824 F.2d at 1146 (1987) (the "Vinyl Chloride" decision). The Vinyl Chloride decision required the Administrator to exercise his judgment under Section 112 in two steps: first, a determination of a "safe" or "acceptable" level of risk considering only health factors, followed by a second step to set a standard that provides an "ample margin of safety," in which costs, feasibility, and other relevant factors in addition to health may be considered.

The four proposed approaches were designed to provide for consideration of a variety of health risk measures and information in the first step analysis under the Vinyl Chloride decision – the determination of "acceptable risk." Included in the alternative approaches were three that consider only a single health risk measure in the first step: (1) Approach B, which considers only total cancer incidence with 1 case per year (case/year) as the limit for acceptability; (2) Approach C, which considers only the maximum individual risk ("MIR") with a limit of 1 in 10 thousand for acceptability; and (3) Approach D, which considers only the maximum individual risk with 1 in 1 million as the limit. The fourth approach, Approach A, was a case-by-case approach that considers all health risk measures, the uncertainties associated with them, and other health information.

In the second step, setting an "ample margin of safety," each of the four approaches would consider all health risk and other information, uncertainties associated with the health estimates, as well as costs, feasibility, and other factors which may be relevant in particular cases. The proposal solicited comment on each of the approaches as well as other approaches for implementing the Vinyl Chloride decision (53 FR 28511-28532). The Agency received many public comments on the approaches from citizen's groups, companies and industry trade groups, State and local governments, and individuals. Most of the comments supported either Approach A or D, with little comment in support of Approach B or C.

*Selection of Approach*

Based on the comments and the record developed in the rulemaking, EPA has selected an approach, based on Approaches A and C but also incorporating consideration of incidence from Approach B and consideration of health protection for the general population on the order of 1 in 1 million from Approach D. Thus, in the first step of the Vinyl Chloride inquiry, EPA will consider the extent of the estimated risk were an individual exposed to the maximum level of a pollutant for a lifetime ("MIR"). The EPA will generally presume that if the risk to that individual is no higher than approximately 1 in 10 thousand, that risk level is considered acceptable and EPA then considers the other health and risk factors to complete an overall judgment on acceptability. The presumptive level provides a benchmark for judging the acceptability of maximum individual risk ("MIR"), but does not constitute a rigid line for making that determination.

The Agency recognizes that consideration of maximum individual risk ("MIR") – the estimated risk of contracting cancer following a lifetime exposure at the maximum, modeled long-term ambient concentration of a pollutant – must take into account the strengths and weaknesses of this measure of risk. It is an estimate of the upperbound of risk based on conservative assumptions, such as continuous exposure for 24 hours per day for 70 years. As such, it does not necessarily reflect the true risk, but displays a conservative risk level which is an upperbound that is unlikely to be exceeded. The Administrator believes that an MIR of approximately 1 in 10 thousand should ordinarily be the upper end of the range of acceptability. As risks increase above this benchmark, they become presumptively less acceptable under section 112, and would be weighed with the other health risk measures and information in making an overall judgment on acceptability. Or, the Agency may find, in a particular case, that a risk that includes MIR less than the presumptively acceptable level is unacceptable in the light of other health risk factors.

In establishing a presumption for MIR, rather than a rigid line for acceptability, the Agency intends to weigh it with a series of other health measures and factors. These include the overall incidence of cancer or other serious health effects within the exposed population, the numbers of persons exposed within each individual lifetime risk range and associated incidence within, typically, a 50 km exposure radius around facilities, the science policy assumptions and estimation uncertainties associated with the risk measures, weight of the scientific evidence for human health effects, other quantified or unquantified health effects, effects due to co-location of facilities, and co-emission of pollutants.

The EPA also considers incidence (the numbers of persons estimated to suffer cancer or other serious health effects as a result of exposure to a pollutant) to be an important measure of the health risk to the exposed population. Incidence measures the extent of health risk to the exposed population as a whole, by providing an estimate of the occurrence of cancer or other serious health effects in the exposed population. The EPA believes that even if the MIR is low, the overall risk may be unacceptable if significant numbers of persons are exposed to a hazardous air pollutant, resulting in a significant estimated incidence. Consideration of this factor would not be reduced to a specific limit or range, such as the 1 case/year limit included in proposed

Approach B, but estimated incidence would be weighed along with other health risk information in judging acceptability.

The limitations of MIR and incidence are put into perspective by considering how these risks are distributed within the exposed population. This information includes both individual risk, including the number of persons exposed within each risk range, as well as the incidence associated with the persons exposed within each risk range. In this manner, the distribution provides an array of information on individual risk and incidence for the exposed population.

Particular attention will also be accorded to the weight of evidence presented in the risk assessment of potential human carcinogenicity or other health effects of a pollutant. While the same numerical risk may be estimated for an exposure to a pollutant judged to be a known human carcinogen, and to a pollutant considered a possible human carcinogen based on limited animal test data, the same weight cannot be accorded to both estimates. In considering the potential public health effects of the two pollutants, the Agency's judgment on acceptability, including the MIR, will be influenced by the greater weight of evidence for the known human carcinogen.

In the Vinyl Chloride decision, the Administrator is directed to determine a "safe" or "acceptable" risk level, based on a judgment of "what risks are acceptable in the world in which we live." 824 F.2d at 1165. To aid in this inquiry, the Agency compiled and presented a "Survey of Societal Risk" in its July 1988 proposal (53 FR 28512-28513). As described there, the survey developed information to place risk estimates in perspective, and to provide background and context for the Administrator's judgment on the acceptability of risks "in the world in which we live." Individual risk levels in the survey ranged from  $10^{-1}$  to  $10^{-7}$  (that is, the lifetime risk of premature death ranged from 1 in 10 to 1 in 10 million), and incidence levels ranged from less than 1 case/year to estimates as high as 5,000 to 20,000 cases/year. The EPA concluded from the survey that no specific factor in isolation could be identified as defining acceptability under all circumstances, and that the acceptability of a risk depends on consideration of a variety of factors and conditions. However, the presumptive level established for MIR of approximately 1 in 10 thousand is within the range for individual risk in the survey, and provides health protection at a level lower than many other risks common "in the world in which we live." And, this presumptive level also comports with many previous health risk decisions by EPA premised on controlling maximum individual risks to approximately 1 in 10 thousand and below.

In today's decision, EPA has selected an approach based on the judgment that the first step judgment on acceptability cannot be reduced to any single factor. The EPA believes that the level of the MIR, the distribution of risks in the exposed population, incidence, the science policy assumptions and uncertainties associated with the risk measures, and the weight of evidence that a pollutant is harmful to health are all important factors to be considered in the acceptability judgment. The EPA concludes that the approach selected best incorporates all of this vital health information, and enables it to weigh them appropriately in making a judgment. In contrast, the single measure Approaches B, C, and D, while providing simple decision making criteria, provide an incomplete set of health information for decisions under section 112. The Administrator believes that the acceptability of risk under section 112 is best judged on the basis of a broad set

of health risk measures and information. As applied in practice, the EPA's approach is more protective of public health than any single factor approach. In the case of the benzene sources regulated here, more than 99 percent of the population living within 50 km would be exposed to risks no greater than approximately 1 in 1 million; and, the total number of cases of death or disease estimated to result would be kept low.

Under the two-step process specified in the Vinyl Chloride decision, the second step determines an "ample margin of safety," the level at which the standard is set. This is the important step of the standard-setting process at which the actual level of public health protection is established. The first step consideration of acceptability is only a starting point for the analysis, in which a floor for the ultimate standard is set. The standard set at the second step is the legally enforceable limit that must be met by a regulated facility.

Even though the risks judged "acceptable" by EPA in the first step of the Vinyl Chloride inquiry are already low, the second step of the inquiry, determining an "ample margin of safety," again includes consideration of all of the health factors, and whether to reduce the risks even further. In the second step, EPA strives to provide protection to the greatest number of persons possible to an individual lifetime risk level no higher than approximately 1 in 1 million. In the ample margin decision, the Agency again considers all of the health risk and other health information considered in the first step. Beyond that information, additional factors relating to the appropriate level of control will also be considered, including costs and economic impacts of controls, technological feasibility, uncertainties, and any other relevant factors. Considering all of these factors, the Agency will establish the standard at a level that provides an ample margin of safety to protect the public health, as required by section 112. Application of this approach to the five source categories under consideration in this rulemaking is summarized in the following discussions.

#### *Maleic Anhydride Process Vents*

Summary of Decision: Benzene is no longer used in the manufacture of maleic anhydride because all plants in the industry have converted their process equipment to the more economical n-butane feed process. Thus, all benzene exposure from this industry has been eliminated, and no Federal regulation is needed. Maleic anhydride plants are, therefore, not discussed in the remaining sections of this notice.

#### *Ethylbenzene/Styrene Process Vents*

Summary of Decision: The existing level of control is judged to provide an ample margin of safety. Under existing State requirements, overall current emissions have been reduced 98 percent or more from uncontrolled levels. The present level of emissions are estimated to present an MIR of 2 in 100 thousand and a total nationwide incidence of about 1 case every 300 years (0.003 case/year). Levels of benzene reported to produce noncancer health effects are at least three orders of magnitude above the exposures comparable to the MIR.

Most people exposed to benzene from these sources are exposed to very low risk levels. Specifically, the risk estimates show: (1) About 600 people are exposed to risk levels of about 1 in 100 thousand reflecting 1 cancer case every 5,000 years (0.0002 case/year) and (2) at least 90 percent of the population modeled to 20 km (about 400,000 people) is exposed to risk levels of less than 1 in 1 million, reflecting about 1 cancer case every 300 years (0.003 case/year). It is anticipated that if modeling were conducted to a 50 km radius, the percentage of the exposed population at risks of less than 1 in 1 million would be at least 99. Further reductions would provide only negligible additional risk and emission reductions (less than 1 percent additional control) and would cost approximately \$0.2 million per year (1982 dollars), which would be about the same in 1988 dollars.

### *Benzene Storage Vessels*

Summary of Decision: In providing an ample margin of safety for this source category, the final standards require effective controls on storage vessels not already controlled. The final standards would reduce nationwide benzene emissions by an estimated additional 20 to 60 percent beyond the baseline level, which already includes emission reductions for most storage vessels. The MIR after application of the standards is estimated to be 3 in 100 thousand. This reflects a reduction from an MIR range of between 4 in 100 thousand and 4 in 10 thousand without the standards. The estimated cancer incidence would be reduced from the range without the standards of 1 case every 10 to 20 years (0.1 to 0.05 case/year) to 1 case every 25 years (0.04 case/year). Levels of benzene reported to produce noncancer health effects are at least three orders of magnitude above the exposure level after an ample margin of safety is provided by EPA.

Most people exposed to benzene from this source category would be exposed to very low levels. The standards are estimated to result in an emission level where: (1) No people are exposed to a risk level greater than 1 in 10 thousand, (2) about 100,000 people would be exposed to a risk level between 3 in 100 thousand and 1 in 1 million, and (3) a majority of the modeled population (70 million people, or greater than 99 percent) is exposed to a risk level of less than 1 in 1 million. While EPA was unable to estimate the cancer incidences associated with various risk levels for this source category, the cancer incidences for the higher risk levels would occur very infrequently and for the lower risk levels would occur about once every 25 years (0.04 case/year). To reduce these exposures further, the next most effective level of control would cost an additional estimated \$1.2 million per year (1982 dollars) or roughly \$1.3 million in 1988 dollars, but it was not chosen because it would not reduce the MIR and would reduce the cancer incidence by only 1 case every 100 years (0.01 case/year).

Summary of the Standards: The final standards require control of all new and existing vessels with capacities greater than or equal to 38 cubic meters (m<sup>3</sup>) (10,000 gallons) used to store benzene. The standards do not apply to storage vessels used for storing benzene at coke by-product recovery facilities because they are considered under the coke by-product recovery plant standards. The standards require use of certain kinds of equipment and work practices for each type of benzene storage vessel. The standards require the use of internal floating roofs (IFR's) with continuous primary seals on fixed roof vessels, and improvements to fittings (e.g.,

gaskets). For external floating roof (EFR) vessels, secondary seals are required. The standards also require periodic inspections of the vessel roofs, seals, and fittings. Detailed summaries of the regulation and changes since proposal are contained in sections IV and V of this notice.

*Coke By-Product Recovery Plants*

Summary of Decision: In providing an ample margin of safety for this source category, the final standards reduce benzene emissions by about 97 percent for affected facilities nationwide. The MIR after application of the standards is estimated to be 2 in 10 thousand and the cancer incidence is about 1 cancer incidence every 20 years (0.05 case/year). This reflects significant risk reduction from the MIR of 7 in 1 thousand and the cancer incidence of 1 cancer incidence every 6 months (about 2 case/year) that are estimated to occur without the standards. Given estimating uncertainties in this case, the MIR level after the standards is comparable to the EPA's benchmark of approximately 1 in 10 thousand. As discussed in Section III of this preamble, EPA views this level as an overstatement of the actual MIR because the emission estimates associated with this level are likely to be overstated. Levels of benzene reported to produce noncancer health effects are at least three orders of magnitude above the exposure level expected after an ample margin of safety is provided by EPA.

Most people exposed to benzene from this source category would be exposed to very low levels. The standards reduce emissions to a level where: (1) Approximately 100 people would be exposed to a risk level between the estimated MIR and about 1 in 10 thousand reflecting about 1 cancer incidence every 5,000 years (0.0002 case/year), (2) about 300,000 people would be exposed to a risk level between 1 in 10 thousand and 1 in 1 million reflecting about 1 cancer incidence every 100 years (0.01 case/year), and (3) a majority of the modeled population (70 million people, or greater than 99 percent) would be exposed to a risk level of less than 1 in 1 million, reflecting about 1 cancer incidence every 25 years (0.04 case/year). To reduce these exposures to the level associated with the next most effective level of control would cost an additional estimated \$6 million per year (1984 dollars), which would be roughly \$6.6 million in 1988 dollars. Furthermore, it would involve the use of a control technology that may not be technically feasible, and would only provide a small overall risk reduction of about 1 percent, reflecting an estimated cancer incidence of 1 in every 33 years (0.03 case/year). Additionally, there would be no change in the MIR of about 2 in 10 thousand.

Summary of Standards: The final standards require that process vessels and tar storage tanks in furnace and foundry coke by-product recovery plants be enclosed and the emissions ducted to an enclosed point in the by-product recovery process where they will be recovered or destroyed. This requirement is based on the use of a gas blanketing system. The same requirements also apply to storage tanks for benzene, benzene-toluene-xylene (BTX) mixtures, and light oil in furnace coke by-product recovery plants. To ensure proper operation and maintenance of the system, the standards require semiannual visual inspections and monitoring to detect and repair leaks as well as annual maintenance inspections. The final standards also require that light-oil sumps be completely enclosed; this requirement is based on the use of a permanent

or removable cover equipped with a gasket. Semiannual visual inspections and monitoring for leak detection and repair are also required for this source.

The final standards establish a zero emissions limit applicable to naphthalene processing, final coolers, and the associated final-cooler cooling towers at both furnace and foundry plants. The limit is based on the use of a wash-oil final cooler, although other types of systems that achieve the emissions limit can also be used.

The final standards also contain provisions for the control of equipment in benzene service, including pumps, valves, exhausters, pressure-relief devices, sampling connections, and open-ended lines. The leak detection and repair requirements are the same as the requirements in 40 CFR 61 subpart V, and additionally include quarterly leak detection and repair requirements for exhausters. A detailed summary of the regulation can be found in section V of this notice.

#### *Benzene Equipment Leaks*

Summary of Decision: The existing standards for this source category (Subpart J of part 61) are judged to provide an ample margin of safety, especially considering the overstatement of emissions. When these standards were issued in 1984, EPA estimated it would reduce emissions by about 70 percent from the level that would occur without the standards. Using these emission estimates (which overstate emissions as discussed in the next paragraph), the MIR was estimated to be 6 in 10 thousand and the incidence was estimated to be 1 case every 5 years (0.2 case/year).

Based on information received in the past year, EPA considers the present level of emissions associated with the existing standards to be substantially lower than previously estimated. Thus the available risk estimates are substantially overstated. The EPA has reached this conclusion after reviewing information demonstrating compliance with the existing standards and new information about emissions from equipment leaks. However, because the changes in the control of equipment leaks, especially leaks of air toxics, and the changes in the analytical tools needed for determining emissions from these sources have occurred very recently, EPA has not been able to develop better estimates of benzene emissions from equipment leaks. If EPA were to roughly estimate emissions based on this information, the resulting MIR would be comparable to the benchmark of approximately 1 in 10,000. (This is discussed further in sections III and IV of this preamble). Levels of benzene reported to produce noncancer health effects are at least three orders of magnitude above current levels of exposure.

Most people exposed to benzene emissions from this source category are exposed to very low risk levels. Even at the estimated emission levels, the existing standards result in: (1) About 1 million people at a level between 1 in 10,000 and 1 in 1 million with an incidence of 1 case every 25 years (0.04 case/year) and (2) the vast majority of the modeled population (200 million people or greater than 99 percent) is exposed at risks of less than 1 in 1 million with an incidence of 1 case every 5 years (0.2 case/year). If the actual emission rates were known, the exposures would be lower than these estimates. To reduce these exposures further to the next most effective level of emission control would require the use of control technologies that may not be technically

feasible at an estimated cost of \$52.4 million per year (1979 dollars), which would be roughly \$75 million in 1988 dollars.

## **II. Background**

### *Regulatory Background*

In 1977, the Administrator announced his decision to list benzene as a hazardous air pollutant under section 112 of the CAA (42 FR 29332, June 8, 1977). Benzene was determined to be a hazardous air pollutant because of its carcinogenic properties, evidenced by elevated leukemia incidence in populations occupationally exposed. Detailed information about the hazard identification, dose/response assessment, exposure assessment and risk characterization for benzene were presented in the preamble to the policy approaches and standards proposed in July 1988 (53 FR 28496), and will not be repeated in today's notice.

The listing of benzene as a hazardous air pollutant was followed by proposal of standards for benzene emissions from maleic anhydride process vents, EB/S process vents, benzene storage vessels, and benzene equipment leaks in 1980 and 1981 (45 FR 26660, April 18, 1980; 45 FR 83448, December 18, 1980; 45 FR 83952, December 19, 1980; and 46 FR 1165, January 5, 1981). On June 6, 1984, after receipt of comments from industry and members of the public, EPA published a final rule setting emission standards for benzene equipment leaks (49 FR 23498) and published proposed standards for benzene emissions from coke by-product recovery plants (49 FR 23522). On that date, EPA also withdrew its proposed standards for maleic anhydride process vents, EB/S process vents, and benzene storage vessels (49 FR 23558). The withdrawal was based on the conclusion that both the benzene health risks to the public from these three source categories, and the potential reductions in health risks achievable with available control techniques were too small to warrant Federal regulatory action under section 112 of the CAA.

On August 3, 1984, the Natural Resources Defense Council (NRDC) filed a petition for review in the United States Court of Appeals for the District of Columbia Circuit, seeking review of the EPA's three withdrawals of proposed benzene emission standards, and the EPA's final standards for benzene equipment leaks (Natural Resources Defense Council, Inc. v. Thomas, No. 84-1387). On October 17, 1984, NRDC petitioned EPA under section 307(d)(7)(B) of the CAA to reconsider its decisions to withdraw standards for maleic anhydride process vents, EB/S process vents, and benzene storage vessels, and to reconsider the promulgated standards for benzene equipment leaks. The EPA denied this petition on August 23, 1985 (50 FR 34144).

On July 28, 1987, the court handed down an en banc decision in a case concerning the national emission standards under Section 112 for vinyl chloride (Docket No. OAQPS 79-3, Part I, Item X-I-4). The court concluded in Vinyl Chloride that EPA had acted improperly in withdrawing a proposed revision to the standards for vinyl chloride by considering costs and technological feasibility without first determining a "safe" or "acceptable" emission level. In light of the Vinyl Chloride opinion, EPA requested a voluntary remand to reconsider its June 6, 1984,

benzene decisions. In an order dated December 8, 1987, the court granted the EPA's motion and established a schedule under which EPA was to propose its action on reconsideration within 180 days of the order and take final action within 360 days of the order. This order was subsequently modified to extend the time for proposal by 45 days and then to establish August 31, 1989, as the deadline for final action. The EPA also decided to reconsider the proposed standards for benzene emissions from coke by-product recovery plants in light of the Vinyl Chloride decision and to publish a supplemental proposal. All of these actions were proposed on July 28, 1988 (53 FR 28496).

#### *Public Participation*

A public hearing was held in Washington, DC, on September 1, 1988, and was attended by about 90 people. Oral testimony was presented by 12 organizations and individuals. The public comment period closed on October 3, 1988, with over 200 comments received among the four dockets. The public comment period was reopened from December 15, 1988, to January 30, 1989, based on the EPA's review of the comments and the number of requests for an extension of the comment period. Additional comments were received, raising the combined number of comments to more than 275.

#### *Legal Framework Under Vinyl Chloride*

The EPA considers the Vinyl Chloride decision to further define the legal framework for setting NESHAP under Section 112 of the CAA. The court set out a two-step process for EPA to follow in making these judgments: first, determine a "safe" or "acceptable risk" level, and then set standards at the level -- which may be equal to or lower, but not higher than, the "safe" or "acceptable" level -- that protects public health with an ample margin of safety. It should be noted that the Vinyl Chloride court acknowledged that EPA could employ a single step analysis under certain circumstances provided cost and feasibility were excluded from consideration. Vinyl Chloride, 824 F.2d at 1165, n.11.

In Vinyl Chloride, the court acknowledged that judgments by EPA concerning scientific uncertainty are a relevant part of the process for establishing NESHAP. As the court noted, Congress, in directing EPA to set NESHAP, recognized that uncertainties over the health effects of the pollutants complicate the task. Vinyl Chloride, 824 F.2d at 1152. These same uncertainties, according to the court, mean that the Administrator's "decision in this area 'will depend to a greater extent upon policy judgments' to which we must accord considerable deference." Id., 824 F.2d at 1162 (citations omitted).

"Safe" or "Acceptable" Level: The first step is for the Administrator to determine what level of risk to health caused by emissions of a hazardous air pollutant is "safe" or "acceptable." (The court used these terms interchangeably.) The court in Vinyl Chloride explicitly declined to determine what risk level is "acceptable" or to set out the method for determining the "acceptable risk" level. Instead, the court stated that these determinations are within the Administrator's discretion.

The court did, however, provide some guidance on the “safe” or “acceptable risk” determination. To make this judgment, “the Administrator must determine what inferences should be drawn from available scientific data and decide what risks are acceptable in the world in which we live.” *Id.*, at 1165. However, the court emphasized that “safe” does not require elimination of all risk. To support these propositions, the court cited *Industrial Union Dept., AFL-CIO v. American Petroleum Inst.*, 448 U.S. 607, 642 (1980) and its statement that “[t]here are many activities that we engage in every day – such as driving a car or even breathing city air – that entail some risk of accident or material health impairment; nevertheless, few people would consider those activities ‘unsafe’.” *Vinyl Chloride*, 824 F.2d at 1165. As a final matter, the court said that the Administrator cannot consider costs or technological feasibility in this step.

**Ample Margin of Safety:** Once an “acceptable risk” level is determined, the second step under *Vinyl Chloride* is to determine whether the emission levels accompanying that determination should be reduced further in providing an “ample margin of safety.” Noting that the purpose of the ample margin of safety requirement is to protect against incompletely understood dangers, uncertainties, and variabilities, the court stated that EPA “may \* \* \* decide to set the level below that previously determined to be safe.” The court reiterated that because the assessment of risk is uncertain, “the Administrator must use his discretion to meet the statutory mandate.” The court added that it is at this stage of the standards-setting process that EPA may consider costs and technological feasibility and other relevant factors: “Because consideration of these factors at this stage is clearly intended to ‘protect the public health,’ it is fully consistent with the Administrator’s mandate under section 112.” *Vinyl Chloride*, 824 F.2d at 1165.

**Uniqueness of Decision:** The effect of the *Vinyl Chloride* decision is to require a decision making process for public health protection decisions unique to section 112, and unlike any other regulatory decision faced by EPA. This is the result of the court’s prescription of two separate steps for decision making, the first in which only health factors can be considered in setting an acceptable risk level, and the second in which additional factors including cost, technological feasibility, and other relevant factors may be considered in providing an ample margin of safety. This scheme is unlike any other under the CAA itself, or any of the other statutes administered by EPA because the acceptable risk that EPA adopts in the first step cannot be exceeded by the standards EPA adopts in the second step. Thus, the EPA’s approach to regulating hazardous air pollutants under section 112 is not applicable to regulatory decisions under other statutes or other sections of the CAA. Regulatory decisions under other statutes or other sections of the CAA will continue to be made using individual deliberative processes pursuant to those distinct statutory mandates.

In contrast to section 112, other EPA statutes have very different structures and legal requirements for decision making on public health standards. For example, while the Safe Drinking Water Act provides for two separate decisions, the first is a purely health-based goal toward which to work, but not necessarily meet; the second is an enforceable standard that is based on cost and feasibility considerations. Under both the Toxic Substances Control Act (TSCA) and the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the balancing of health concerns and benefits of continued chemical use, and control costs are explicitly provided

for in decision making. The Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act both require statutory decision making very different from the bifurcated process mandated by the court for Section 112.

Prior to issuance of Vinyl Chloride decision by the DC Circuit Court, the EPA's recent judgments under section 112 were made in integrated approaches that considered a range of health and risk factors, as well as cost and feasibility in certain cases. However, the Vinyl Chloride decision has required a change in the EPA's approach to section 112, since the previously employed integrated approaches did not partition consideration of health factors into a first step separate from consideration of the other relevant factors. Thus, the Vinyl Chloride decision requires EPA to consider whether a risk is acceptable without at the same time considering benefits of the activity causing risk, feasibility of control, or other factors that EPA (or anyone) would normally consider in determining whether a risk was "acceptable."

### **III. Application of Policy to Benzene Source Categories**

#### *Introduction*

This section of the preamble explains the application of the EPA's policy for the regulation of the benzene source categories discussed in the July 28, 1988, proposal (53 FR 28496). For each source category, the following are provided: (1) Background information particularly noting any changes to the EPA's risk assessment since the July 1988 proposal, (2) the decision on the acceptable risk noting the health-related factors and uncertainties associated with the EPA's decision, and (3) the decision on the ample margin of safety noting health-related impacts, technological feasibility, and cost information associated with this decision. For those sources for which EPA made decisions that result in additional regulatory requirements, the requirements are explained in Section V of this notice.

#### *Ethylbenzene/Styrene Process Vents*

**Background:** This source category covers process vents of plants manufacturing ethylbenzene, styrene, or both. (Benzene emissions from equipment leaks and storage vessels at EB/S plants have been considered separately and are not included in this source category). As of 1985, there were 13 plants in this source category. Information received during the public comment period indicates that emissions have declined since 1985 and emissions are now estimated to be 135 megagrams per year (Mg/yr) or less.

**Decision on Acceptable Risk:** The baseline MIR of  $2 \times 10^{-5}$  is below the presumptive benchmark of approximately  $1 \times 10^{-4}$  (which is 1 in 10 thousand expressed in scientific notation). In estimating these risk levels, EPA has not found that co-location of EB/S plants significantly influences the magnitude of the MIR or other risk levels. The nationwide incidence of cancer from exposure to emissions from these facilities is estimated to be about 1 case every 330 years (0.003 case/year) or lower. The majority (more than 90 percent) of the population within 20 km of these

sources is exposed to risk levels lower than  $1 \times 10^{-6}$ . For exposures to risk levels greater than  $1 \times 10^{-6}$ , the incidence is estimated to be 1 case every 10,000 years (0.0001 case/year). Benzene concentrations reported to produce noncancer health effects are at least three orders of magnitude above the exposures predicted from these sources. After considering all these factors, EPA judged the emission level associated with an MIR of  $2 \times 10^{-5}$  is acceptable.

**Decision on Ample Margin of Safety:** The EPA considered selecting a control level more stringent than the level associated with the acceptable risks. This option would require control of the few remaining uncontrolled intermittent emission sources using 98-percent efficient combustion devices (e.g., boilers and flares). In comparing this control option and the existing level of control, EPA found that they provide essentially the same level of safety. Both control levels reflect a significant reduction in risks and emissions from the uncontrolled level. Control of these sources would further reduce benzene emissions by approximately 70 to 90 Mg/yr at most and would reduce the estimated MIR from  $2 \times 10^{-5}$  to  $1 \times 10^{-5}$ . The annual incidence would be reduced by about 1 case every 500 years (0.002 case/year).

The number of people exposed at risks greater than  $1 \times 10^{-6}$  is essentially the same between these two control levels. For the total population exposed to these sources, the incidence would change from 1 case every 330 years (0.003 case/year) to 1 case every 1,000 years (0.001 case/year). Essentially all (95 percent) of this additional reduction in incidence occurs in the population exposed to risks lower than  $1 \times 10^{-6}$ . The proportion of the population at risk levels below  $1 \times 10^{-6}$  is not changed by this emission reduction. In addition, benzene concentrations reported to produce noncancer health effects are at least three orders of magnitude above the exposures predicted for these sources.

As noted above, this control option will reduce benzene emissions by 70 to 90 Mg/yr, which represents less than an additional 1 percent reduction over the uncontrolled level. The cost of this additional emission reduction (and consequent risk reduction) would be about \$200,000/yr (1982 dollars). While this additional cost is small, it is disproportionately large in comparison to the small additional emission and risk reduction achieved.

After considering all of these factors, EPA judged that the existing level of controls provides an ample margin of safety. In addition, EPA decided not to set standards to mandate the existing level of controls. Existing controls in the EB/S industry are in the form of product recovery devices or the routing of emissions to the process unit's boilers or other boilers onsite to conserve energy (less fuel would be required due to the energy content of the waste stream). Thus, there is no incentive for removal of existing controls.

Additionally, there is no incentive for new sources to waste product or energy, and major new sources would be subject to other EPA requirements (e.g., new source review [NSR], prevention of significant deterioration [PSD]). Thus, less effective controls are not expected in the future. For these reasons, EPA has concluded that Federal standards mandating these controls are not warranted.

*Benzene Storage Vessels*

Background: This source category covers vessels used to store benzene. These vessels are typically located at petroleum refineries, chemical plants, and bulk storage terminals. As of 1984, 126 facilities with benzene storage vessels had been identified. As noted in the July 28, 1988, Federal Register notice, nationwide baseline (i.e., no NESHAP) emissions from benzene storage vessels are estimated to be about 620 to 1,290 Mg/yr. The range of emissions reflects uncertainty about the presence of shingled seals versus continuous seals on existing vessels with IFR's; the lower end of this range reflects the assumption that all storage vessels have continuous seals, while the upper end is based on the assumption that some vessels (17 percent of the existing IFR vessels) are equipped with shingled seals, which emit more benzene than continuous seals. The baseline incidence associated with these emission estimates is estimated to be 1 case every 10 to 20 years (0.1 to 0.05 case/year). The baseline MIR ranges from  $4 \times 10^{-5}$  to  $4 \times 10^{-4}$ .

Decision on Acceptable Risk: The baseline MIR ( $4 \times 10^{-5}$  to  $4 \times 10^{-4}$ ), while ranging above the presumptive risk of approximately  $1 \times 10^{-4}$ , is judged to be within the acceptable range after consideration of the following factors.

First, the upper end of the range ( $4 \times 10^{-4}$ ) is very likely an overestimate of the MIR because it assumes that all storage vessels have shingled seals at the plants that would also have the highest MIR's if all vessels in the industry had continuous seals. Based on information received from industry in 1978, EPA estimated that 12 percent of the nationwide benzene storage capacity was in vessels with shingled seals. This was estimated to be only about 17 percent of the existing IFR vessels that store benzene. The EPA believes that shingled seals have not been installed on new vessels for the past several years as general industry practice. Accordingly, the number of vessels equipped with shingled seals is decreasing over time; consequently the associated risk is also decreasing as existing vessels are replaced by new vessels. Therefore, the assumption that all vessels in the worst-case plant have shingled seals for the upper end of the MIR range is a unique conservative assumption for this source category. In addition, the emission estimate for storage vessels equipped with shingled seals is overstated for the following reason. The only test series of IFR vessels with shingled seals had testing irregularities, resulting in inaccurately high emission estimates. These test irregularities are described in detail in the EPA document "Benzene Emissions from Benzene Storage Tanks -- Background Information for Proposal to Withdraw Proposed Standards" (EPA-450/3-84-004, March 1984). Because there is no way to determine the proportion of emissions attributable to the use of shingled seals versus the test methodology, the emission estimate for shingled-seal vessels continues to reflect all the uncertainty from that test series (49 FR 23563, June 6, 1984). While EPA is unable to quantify these uncertainties, EPA qualitatively considered the effect of these uncertainties (as well as other uncertainties in its risk assessment) in its judgment of acceptability.

Second, even if the MIR were not overestimated, EPA estimated that only 10 people (out of the total modeled population of 70 million) are at risks greater than or equal to  $1 \times 10^{-4}$ , and virtually no cancer incidence is associated with this risk level. In estimating these risk levels, EPA has not found that co-location of plants significantly influences the magnitude of the MIR or other

risk levels. Where two or more of the model plants used for the analysis might occur at one site (e.g., both a producer and a consumer of benzene), the risks were calculated from their total emissions. In addition, EPA estimated that the majority of the people (about 99 percent) exposed to benzene from this source category would be exposed to a risk level of less than  $1 \times 10^{-6}$ , reflecting 1 cancer incidence every 12 years (0.08 case/year), and that 900,000 people would be exposed at a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$ , reflecting 1 cancer incidence every 50 years (0.02 case/year). The baseline incidence is estimated to be 1 incidence every 10 to 20 years (0.1 to 0.05 cancer case/year). This range reflects the range of emission estimates (620 to 1,290 Mg/yr). Virtually all of the incidence is associated with the population at a risk of less than  $1 \times 10^{-5}$ . Thus, even though one end of the range of the EPA's MIR estimate for this source category is above  $1 \times 10^{-4}$ , it is important to consider that almost all of the exposure to benzene from storage vessels is associated with risks well below the benchmark of approximately  $1 \times 10^{-4}$ .

The EPA also considered the noncancer health effects associated with benzene exposures at levels comparable to the baseline MIR range. Noncancer health effects have been associated with exposure to benzene, but the levels reported to produce such effects are two to three orders of magnitude above exposures comparable to the MIR range of  $4 \times 10^{-5}$  to  $4 \times 10^{-4}$ , especially with the likely overstatement of the top end of the range.

After considering all these factors, EPA judged that the baseline emission level is acceptable.

**Decision on Ample Margin of Safety:** The EPA considered selecting a level of emissions more stringent than the level associated with acceptable risk in providing an ample margin of safety for this source category. This would require all vessels to have emission reduction equipment that many vessels already have. Specifically, it would require the use of an IFR with continuous primary seals on each existing fixed roof vessel, and more effective continuous primary seals on any new vessel with an IFR. It would also require improvements to fittings (e.g., gaskets) on the roofs of all IFR vessels. On each vessel with an EFR, this option would require secondary seals. These are similar controls to those that are required for volatile organic liquid (VOL) storage vessels (including benzene vessels) in 40 CFR 60 Subpart Kb, which affects vessels constructed or rebuilt after July 23, 1984. This level of control was labeled Option 2 in the July 28, 1988, proposal (53 FR 28496).

Control Option 2 would reduce the estimated MIR to  $3 \times 10^{-5}$  from the baseline range of  $4 \times 10^{-5}$  to  $4 \times 10^{-4}$ . Because no facility could have vessels with shingled seals, which represent the upper end of the baseline range, all vessels would be required to have continuous seals under the control option and the risks are not expressed as a range. Thus, no one would be potentially exposed to a risk of greater than or equal to  $1 \times 10^{-4}$ . The number of people estimated to be exposed to a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$  would be reduced from 900,000 at baseline to 100,000 with this control option. The majority of the modeled exposed population (greater than 99 percent) would be exposed to a risk level less than  $1 \times 10^{-6}$  with Option 2. While EPA was unable to estimate the cancer incidences associated with various risk levels after control to this option for this source category, the cancer incidences for the higher risk levels would occur

infrequently, and for the lower levels would occur about once every 25 years (0.04 case/year). Overall, the total nationwide incidence would be reduced from a range of 1 incidence every 10 to 20 years (0.1 to 0.05 case/year) to 1 incidence every 25 years (0.04 case/year). In addition, levels of benzene reported to produce noncancer health effects are at least three orders of magnitude above the levels expected under Option 2.

Control Option 2 would reduce benzene emissions by a range between 20 to 60 percent (110 to 780 Mg/yr) in comparison to the emissions without standards. To achieve this emission reduction (and consequent risk reduction) would cost \$0.1 million/yr (1982 dollars). This cost is considered to be relatively small.

The EPA also considered a more stringent control level, which would require the controls in Option 2 and additionally require secondary seals for IFR vessels (Option 1 in the July 28, 1988, proposal notice, 53 FR 28496). This additional control would not result in any additional reduction in the MIR beyond that achieved by Option 2. The number of people estimated to be exposed to a risk level greater than  $1 \times 10^{-6}$  is estimated to be reduced from 100,000 (Option 2) to 80,000 (Option 1). In both cases, the vast majority of the exposed population (greater than 99 percent) is at a risk of less than  $1 \times 10^{-6}$ . Overall, the total nationwide incidence would only be reduced from 1 incidence every 25 years (0.04 case/year) for Option 2 to 1 incidence every 33 years (0.03 case/year) for Option 1. This additional incidence reduction is associated mainly with the population exposed to risk levels below  $1 \times 10^{-6}$ . Levels of exposure reported to produce noncancer health effects are at least three orders of magnitude above the levels of exposure expected for Option 1, just as for Option 2. The additional cost of Option 1 over Option 2 would be \$1.2 million/yr (1982 dollars).

Based on the factors discussed above, EPA decided that the level of control reflected by Option 2 provides an ample margin of safety. Although the emissions associated with the baseline risks are considered to be acceptable, they can be reduced further, achieving additional risk reductions, at a reasonable cost using the control technology included in Option 2. Selecting Option 2 also ensures that any existing shingled seals are replaced with continuous seals, thus addressing one of the uncertainties associated with the EPA's risk assessment. In addition, EPA concluded that additional controls beyond Option 2 are not warranted. The costs of additional controls beyond Option 2 are disproportionately high considering the small reductions in risk and incidence which are achievable.

#### *Coke By-Product Recovery Plants*

Background: The risk analysis was revised after the July 1988 proposal based on comments that the industry's operating status should be updated. There are now 36 coke by-product recovery plants. The nationwide baseline benzene emissions are estimated to be 17,000 Mg/yr. The revised baseline estimates of health risk indicate an MIR of  $7 \times 10^{-3}$  and an annual cancer incidence of 1 case every 6 months (2 cases/year). More information regarding the updated estimates can be found in Section IV of this preamble and in the BID.

Decision on Acceptable Risk: The baseline risk of  $7 \times 10^{-3}$  is unacceptable for benzene, a known human carcinogen. In considering the decision on acceptable risk for this source category, EPA focused on control to a level that would result in an estimated MIR of  $2 \times 10^{-4}$ . The EPA considers this MIR to be in the acceptable range after considering several factors.

First, the long-term emissions and, therefore, the MIR are likely to be overstated because EPA assumed that coke batteries operate at full capacity for 70 years. In fact, presently not all plants are continuously operating at full capacity (including some of the plants with the highest risks). In addition, the decline in the domestic coke industry makes it likely that the EPA's estimate overstates the long-term emissions. There is considerable uncertainty in predicting the utilization of coke batteries. Therefore, EPA made the assumption of full capacity for 70 years, recognizing the effect of this assumption (as well as other assumptions) on its risk assessment. Thus, EPA believes the MIR is not likely to be much different than the benchmark of approximately  $1 \times 10^{-4}$  even though EPA is unable to quantify these uncertainties and, therefore, adjust the MIR for this source category. However, EPA considered this likely overestimation qualitatively in its judgment of acceptability. Furthermore, over time, the residual emissions from one group of sources in this category (equipment leaks) may decrease as operators use better equipment (e.g., improved valve packing) in addition to the required work practice program.

Second, EPA estimated that 100 people (out of the total modeled population of 70 million) potentially would be exposed to risks of  $1 \times 10^{-4}$  or greater, with 1 cancer incidence every 5,000 years among this group of 100 people (0.0002 case/year). In estimating these risk levels, EPA has not found that co-location of coke by-product recovery plants significantly influences the magnitude of the MIR or other risk levels. In addition, EPA estimated that the vast majority of the modeled population (greater than 99 percent) exposed to benzene from this source category would be exposed to a risk level of less than  $1 \times 10^{-6}$  reflecting 1 cancer incidence every 25 years (0.04 case/year), and that 300,000 people would be exposed at a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$  reflecting 1 cancer incidence every 100 years (0.01 case/year). Of the total cancer incidence (1 cancer incidence every 20 years, i.e., 0.05 case/year), 80 percent is associated with the large population at risks of less than  $1 \times 10^{-6}$ . Thus, even though EPA estimates an MIR of about  $2 \times 10^{-4}$  for this option, it is important to consider that almost all the exposure to benzene from this source category is associated with risks well below the benchmark of approximately  $1 \times 10^{-4}$ .

The EPA also considered the noncancer health effects associated with benzene exposures at levels comparable to an MIR level of  $2 \times 10^{-4}$ . Noncancer health effects have been associated with exposure to benzene, but the probability is unlikely of the effects occurring at exposures comparable to an MIR level of  $2 \times 10^{-4}$ . Levels of benzene reported to produce such effects are three orders of magnitude higher than the concentrations comparable to an MIR of  $2 \times 10^{-4}$ .

After considering all these factors, EPA judged the emission level associated with an MIR of  $2 \times 10^{-4}$  to be acceptable.

Decision on Ample Margin of Safety: The EPA considered selecting a level of emissions more stringent than the level associated with acceptable risks in providing an ample margin of safety for this source category. This option (Option 1) would require additional control over the acceptable risk level (Option 2) of storage vessels at foundry coke by-product recovery plants and would also require use of dual mechanical seals on pumps and sealed bellows valves (i.e., assumed to be 100 percent control) at both furnace and foundry coke by-product recovery plants. The control technologies and their estimated impacts are presented for each emission point in Table 1 for Options 1 and 2. It should be noted that EPA has not concluded that leakless valves/sealed bellows valves will always effectively eliminate emissions or that they are available for all sizes and types of equipment in benzene service. Nevertheless, EPA evaluated Option 1 to determine if it should be selected to reflect an ample margin of safety even though there would be technological feasibility issues in implementing this option.

**Table 1 – Controls Included in Each Option<sup>a</sup>**

Emission points	Control technology efficiency (%)	Option 1		Option 2	
		Furnace	Foundry	Furnace	Foundry
Final cooler, cooling tower; naphthalene processing/handling	Wash-oil final cooler (100)	X	X	X	X
Tar decanter, tar intercepting sump and flushing-liquor circulation tank	Gas blanketing (98 <sup>b</sup> )	X	X	X	X
Tar storage and tar-dewatering tanks	Gas blanketing (98)	X	X	X	X
Light-oil condenser, light-oil decanter, wash-oil decanter, and wash-oil circulation tanks	Gas blanketing (98)	X	X	X	X
Excess ammonia-liquor storage tank	Gas blanketing (98)	X	X	X	
Light-oil and BTX storage tanks	Gas blanketing (98)	X	X	X	
Benzene storage tanks	N <sub>2</sub> gas blanketing (98)	X	X	X	
Light-oil sump	Cover (98)	X	X	X	X
Pumps	Monthly inspections (83)			X	X
	Dual mechanical seals (100)	X	X		
Valves	Monthly inspections (73)			X	X
	Sealed-bellows valves (100)	X	X		
Exhausters	Quarterly inspections (55)			X	X
	Degassing reservoir vents (100)	X	X		
Pressure-relief devices	Rupture disc system (100)	X	X	X	X
Sampling connection systems	Closed-purge sampling (100)	X	X	X	X
Open-ended lines	Cap or plug (100)	X	X	X	X

<sup>a</sup> The control options analyzed to determine an ample margin of safety are the same as those analyzed for the July 1988 proposal (53 FR 28496), except that control options less stringent than Option 2, the level determined to be in the acceptable range, are not shown on the table. The impacts associated with these control options have been revised since the July 1988 proposal to reflect updated information on the industry operating status. These revisions are explained in greater detail in Section 6 of the BID.

<sup>b</sup> 95-percent efficiency for tar decanter.

In comparing Options 1 and 2, EPA found that they provide essentially the same level of safety. Each reflects significant risk reduction in comparison to the baseline risks. Although the estimated number of people exposed to a risk level greater than or equal to  $1 \times 10^{-4}$  would be reduced from 100 to 50 under Option 1, EPA estimates that Option 1 would not reduce the MIR below the Option 2 level of  $2 \times 10^{-4}$ . The number of people exposed to a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$  would be reduced from 300,000 to 200,000 under Option 1. Under both options, the vast majority of the exposed population (greater than 99 percent) would be at risk levels of less than  $1 \times 10^{-6}$ . For the population exposed to a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$ , the incidence would change from 1 case every 100 years (0.01 case/year) under Option 2 to 1 case every 140 years (0.007 case/year) under Option 1; for the population exposed to risks below  $1 \times 10^{-6}$ , the incidence would change only from 1 case every 25 years (0.04 case/year) under Option 2 to 1 case every 33 years (0.03 case/year) under Option 1. Overall, the total nationwide incidence would be reduced from 1 case every 20 years (0.05 case/year) to 1 case every 33 years (0.03 case/year) or only by an additional 0.02 case/year. Most (about 80 percent) of this additional reduction in incidence in Option 1 compared to Option 2 occurs in the population exposed to risks in the  $1 \times 10^{-6}$  range or lower. In addition, levels reported to produce noncancer health effects are about three orders of magnitude above levels expected under either option.

Option 1 reduces benzene emissions by about 98 percent, whereas Option 2 reduces benzene emissions by about 97 percent in comparison to the emissions that would occur without the standards. This reflects only an additional 1 percent reduction for Option 1. Also, the relative difference between these options may be even smaller than estimated. This is due to the uncertainty that sealed bellows valves would actually achieve the assumed 100 percent reduction in Option 1 and the potential for higher emission reduction than estimated for the equipment leak detection and repair program under Option 2. To achieve this emission reduction (and consequent risk reduction), Option 1 would increase the annualized cost by about \$6 million/yr (1984 dollars). While this additional cost is relatively small overall, it is disproportionately large in comparison to the small additional emission and health risk reductions associated with Option 1 in comparison to Option 2.

In conclusion, EPA decided that Option 2 provides an ample margin of safety. The EPA judged the risk reductions for Options 1 and 2 to be essentially the same and the greater control cost of Option 1 to be high in relation to the small additional emission and risk reduction achieved. In doing so, EPA considered the likely overstatement of long-term emissions and risks and the question of technical feasibility.

### *Benzene Equipment Leaks*

Background: This source category covers emissions of benzene from pieces of equipment handling process streams that contain greater than 10 percent benzene, by weight. These equipment pieces include pumps, pipeline valves, open-ended valves, flanges, compressors, pressure-relief valves, sampling connections, process drains, and product accumulator vessels. In 1984, there were an estimated 131 facilities in this source category.

When Subpart J of Part 61, the benzene equipment leaks NESHAP, was promulgated in 1984, EPA estimated that this regulation would reduce emissions from about 7,900 Mg/yr to 2,500 Mg/yr (a 69 percent reduction). As noted in the July 28, 1988, Federal Register notice, EPA viewed the estimate of 2,500 Mg/yr for current emissions as being an upperbound estimate, and recognized that actual emissions may be substantially lower. The EPA reached this conclusion after reviewing compliance report information from facilities subject to the existing standards and other information for facilities handling toxic compounds. Information obtained since proposal has further substantiated this conclusion. The basis for this conclusion is summarized below and is discussed in more detail in section IV and in the BID.

During the consideration of the public comments, EPA examined compliance reports from 1987 and 1988 for a randomly-selected sample of 25 facilities subject to the benzene NESHAP. This review showed many facilities had no leaking valves or pumps (0.0 percent) and no facilities had more than 1.5 percent leaking valves. The average leak rate for valves was 0.27 percent. This performance is better than an average expected leak rate of about 3 to 5 percent. In addition to the compliance reports, EPA also reviewed a limited amount of comprehensive data for a few process units with equipment in benzene service. These data show emission rates a factor of 20 to 30 below levels predicted by the earlier EPA studies. However, these more recent results do not provide a basis for developing new emission factors that would be generally applicable to all facilities. To rederive the emission estimates will require additional information and analysis of current industry practices. As this information has been received only recently, EPA has not been able to conduct the necessary studies and analyses in time to revise the emission estimates for benzene equipment leaks. The EPA has initiated a negotiated rulemaking to develop a new regulatory approach that will result in quantifiable emission levels, give credit for good original plant design, and motivate innovation (54 FR 17944, April 25, 1989). This effort is expected to require at least 6 months to complete. Consequently, the emission and risk estimates remain essentially as presented in the July 28, 1988, Federal Register notice.

Decision on Acceptable Risk: Based on 1984 emission estimates, the MIR is estimated to be  $6 \times 10^{-4}$ . However, as discussed previously under "Background" (and as discussed in detail in section IV, in response to comments), EPA considers the emission estimates to be overstated by roughly a factor of 5 to 20, or more. If actual emissions could be quantified and modeled in the exposure analysis, the risk estimates would decrease proportionately to the emissions, and would be comparable to the presumptive risk benchmark. An additional factor in this overstatement of emissions is that the analysis was developed assuming facilities continued to operate at the estimated emission rate for 70 years. However, EPA expects that, over time, emissions may continue to decrease due to improved control of air toxics through use of better design, operation, and maintenance of facilities. Given all these factors, EPA concludes that the MIR for this category is more likely to be less than the benchmark of approximately  $1 \times 10^{-4}$ , and will use this in its judgment on acceptability.

The estimated annual cancer incidence (based on the overstated emission estimates) is 1 case every 5 years (0.2 case/year) in a total modeled population of 200 million. The estimated incidence among the 2,000 people predicted to be at lifetime risks greater than  $1 \times 10^{-4}$  is only 1

case every 200 years (0.005 case/year). In estimating these risk levels, EPA has not found that co-location of facilities significantly influences the magnitude of the MIR. In addition, EPA estimated the majority of the population (greater than 99 percent) exposed to benzene from this source category would be exposed to risk levels below  $1 \times 10^{-6}$ . The incidence predicted for the population exposed to risks smaller than  $1 \times 10^{-6}$  is 1 case every 5 years (0.2 case/year), and the incidence for the population exposed to risks greater than  $1 \times 10^{-6}$  is 1 case every 20 years (0.05 case/year).

The EPA also considered the noncancer health effects associated with benzene exposures at current levels of exposure from this source category. Benzene concentrations reported to produce noncancer health effects are two to three orders of magnitude above the exposures predicted for these sources.

After considering all of these factors, especially the substantial overstatement of emissions, EPA judged that the present, controlled level of emissions and risks are acceptable.

**Decision on Ample Margin of Safety:** The EPA considered selecting a level of emissions more stringent than the level associated with the existing standards. The additional control of Option 1 reflects the use of dual mechanical seals for pumps, and sealed bellows valves. For the purpose of this analysis, this equipment is considered to be leakless (i.e., 100 percent control). However, it is not known if leakless valves/sealed bellows valves will effectively eliminate emissions or if they are available for all sizes and types of equipment in benzene service. Thus, it should be noted that EPA has not concluded that leakless valves/sealed bellows valves will effectively eliminate leaks. Information is needed on the magnitude of emissions released when a sealed bellows valve fails, failure rates of these valves, and appropriate procedures for monitoring valves for failures before any conclusions are made. In addition, a better understanding of the factors affecting equipment leaks and development of new regulatory approaches is needed before significant further reductions in exposures will be assured. Nevertheless, EPA considered Option 1 to determine if it should be selected to provide an ample margin of safety even though there would be technological feasibility issues in implementing this option.

Under Option 1, the estimated MIR would be reduced by roughly a factor of three, and the nationwide incidence would be reduced from 1 case every 5 years (0.2 case/year) under the current NESHAP baseline to 1 case every 10 years (0.1 case/year). As discussed under the "Decision on Acceptable Risk," EPA views the estimate of the MIR for this source category as significantly overstated. The number of people exposed to a risk level between  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$  would be reduced from about 1 million to 300,000 under Option 1. For the people exposed to these risk levels, the incidence would change from 1 case every 200 years (0.005 case/year) to 1 case every 1,000 years (0.001 case/year) and from 1 case every 25 years (0.04 case/year) to 1 case every 100 years (0.01 case/year), respectively. The number exposed to a risk level less than  $1 \times 10^{-6}$  would be the same under Option 1 and the existing standards, with more than 99.5 percent of the total population of 200 million exposed to these risk levels. Most (about 90 percent) of the additional reduction in incidence in Option 1 compared to the existing standards would occur in the population exposed to risks in the  $1 \times 10^{-6}$  range or lower. In addition,

benzene concentrations reported to produce noncancer health effects are at least two to three orders of magnitude above the concentrations expected under Option 1 or the existing standards.

Option 1 is estimated to reduce benzene emissions by about 50 percent from the level of the standards. The relative difference between the two control levels may be substantially smaller than this estimate. This is due to the uncertainty that sealed bellows valves would actually achieve the assumed 100 percent reduction in Option 1 and the greater than predicted reductions observed with the current standards' leak detection and repair program. Because of the large uncertainty in the emission levels under the current standards, the likely additional emission reduction cannot be estimated. Implementation of the requirements of Option 1 would increase the annualized control cost by \$52.4 million/yr (1979 dollars). (Docket No. A-79-27, Item V-A-1). The majority of the estimated cost is from the cost of sealed bellows valves.

Although Option 1 shows some additional emission and risk reduction may be achievable, the control cost is disproportionately large when compared to the small reductions in risk which could be achieved. If the actual emission reduction were known and used, the option would likely be even less effective. Recognizing the uncertain bias in the emission estimates, the large proportion of the incidence associated with lifetime risks less than  $1 \times 10^{-6}$ , the questions regarding technical feasibility, and the costs of additional controls, EPA judged the emission levels associated with the existing NESHAP to protect public health with an ample margin of safety. Therefore, additional control beyond the existing NESHAP is not warranted and will not be required.

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## **Appendix C**

### **Schedule for Source Category MACT Standards**

**Exhibit C.1**  
**EPA - Clean Air Act - Title III**  
**2-Year MACT Standards**

<b>MACT Standard / Source Categories</b>	<b>Number of Source Categories</b>	<b>CFR Subparts</b>	<b>Statutory Date</b>	<b>Administrator Signed Promulgation</b>	<b>Fed Register Publication &amp; Citation</b>	<b>Initial Compliance Date</b>
DRY CLEANING	5	M	11/15/92	09/13/93	09/22/93 (58FR49354)	12/20/93
Commercial dry cleaning dry-to-dry						
Commercial drycleaning transfer machines*						
Commercial drycleaning transfer machines						
Industrial drycleaning dry-to-dry						
Industrial drycleaning transfer machines						
HAZARDOUS ORGANIC NESHAP	1	F, G, H, I	11/15/92	02/28/94	04/22/94 (59FR19402)	10/24/94

Key Legend:

\* = denotes area source category

Admin signed date = actual date EPA Administrator signed package

**Exhibit C.2**  
**EPA - Clean Air Act - Title III**  
**4-Year MACT Standards**

<b>MACT Standard / Source Categories</b>	<b>Number of Source Category</b>	<b>CFR Subparts</b>	<b>Statutory Date</b>	<b>Administrator Signed Promulgation</b>	<b>Fed Register Publication &amp; Citation</b>	<b>Initial Compliance Date</b>
AEROSPACE INDUSTRY	1	GG	11/15/94	07/31/95	09/01/95 (60FR45948)	09/01/98
ASBESTOS (delisted)	1	--	11/15/94	11/14/95	11/30/95 (60FR61550)	11/30/95
CHROMIUM ELECTROPLATING	6	N	11/15/94	11/22/94	01/25/95 (60FR49848)	01/25/96 decor; 01/25/97 others
Chromic Acid Anodizing						
Chromic Acid Anodizing*						
Decorative Chromium Electroplating						
Decorative Chromium Electroplating*						
Hard Chromium Electroplating						
Hard Chromium Electroplating*						
COKE OVENS	1	L	12/31/92	10/23/93	10/27/93 (58FR57898)	11/15/93
COMMERCIAL STERILIZERS		2	O	11/23/94	11/22/94	12/06/94 (59FR62585)
Commercial Sterilization Facilities						
Commercial Sterilization Facilities*						
DEGREASE ORGANIC CLEANERS	2	T	11/15/94	11/15/94	12/02/94 (59FR61801)	12/02/97
Halogenated Solvent Cleaners						
Halogenated Solvent Cleaners*						
INDUSTRIAL COOLING TOWERS	1	Q	11/15/94	07/30/94	09/08/94 (59FR46339)	03/08/96
MAGNETIC TAPE	1	EE	11/15/94	11/22/94	12/15/94 (59FR64580)	12/15/96
MARINE VESSELS	1	Y	11/15/94	07/28/95	09/19/95 (60FR48388)	09/19/99
OFF-SITE WASTE TREATMENT	1	DD	11/15/94	05/28/96	07/01/96 (61FR34139)	07/01/99
PETRO REFINERIES	1	CC	11/15/94	07/28/95	08/18/95 (60FR4344)	08/18/98

**Exhibit C.2 (cont.)  
EPA - Clean Air Act - Title III  
4-Year MACT Standards**

MACT Standard / Source Categories	Number of Source Category	CFR Subparts	Statutory Date	Administrator Signed Promulgation	Fed Register Publication & Citation	Initial Compliance Date
PRINTING/PUBLISHING	1	KK	11/15/94	05/15/96	05/30/96 (61FR27132)	05/30/99
POLYMERS & RESINS I	9	U	11/15/94	07/15/96	09/05/96 (61FR46906)	03/05/97
Butyl Rubber						
Epichlorohydrin Elastomers						
Ethylene Propylene Rubber						
Hypalon (TM) Production						
Neoprene Production						
Nitrile Butadiene Rubber						
Polybutadiene Rubber						
Polysulfide Rubber						
Styrene-Butadiene Rubber & Latex						
POLYMERS & RESINS II	2	W	11/15/94	02/28/95	03/08/95 (60FR12670)	03/03/98
Epoxy Resins Production						
Non-Nylon Polyamides Production						
POLYMERS & RESINS IV	6	JJJ	11/15/94	05/15/96	09/12/96 (61FR48208)	03/12/97
~Acrylonitrile-Butadiene-Styrene						
~Methyl Methacrylate-Acrylonitrile+						
Methyl Methacrylate-Butadiene++						
~Polystyrene						
Styrene Acrylonitrile						
Polyethylene Terephthalate						
SECONDARY LEAD SMELTERS	1	X	11/15/94	5/31/95	06/23/95 (60FR32587)	06/23/97
SHIPBUILDING MACT	1	II	11/15/94	11/14/95	12/15/95 (60FR64330)	12/16/97
STAGE I GASOLINE DISTRIBUTION	1	R	11/15/94	11/23/94	12/14/94 (59FR64303)	12/15/97
WOOD FURNITURE	1	JJ	11/15/94	11/14/95	12/07/95 (60FR62930)	11/21/97
total sources	40					

**Exhibit C.2 (cont.)  
EPA - Clean Air Act - Title III  
4-Year MACT Standards**

Table Legend:

\* area source categories

+ Methyl Methacrylate-Acrylonitrile-Butadiene-Styrene

++ Methyl Methacrylate-Butadiene-Styrene Terpolymers

Admin signed date = actual date EPA Administrator signed package

**Exhibit C.3  
EPA - Clean Air Act - Title III  
7-Year MACT Standards**

Statutory date - 11/15/97 (42 Source Categories)  
Updated as of January 1998

<b>7 YEAR STANDARDS</b>	<b>PROPOSE</b>	<b>PROMULGATE</b>
Agriculture Chemicals Production (10)*	10/27/97	03/99
Acrylic/Modacrylic Fibers (GMACT)	12/97	12/98
Manuf. of Tetrahydrobenzaldehyde^^	08/15/97	02/98
Chlorine Manuf.	11/99	11/2000
Chromium Chemicals Manuf.	--	delisted 5/17/96
Cyanide Chemicals Production (3)*	11/99	11/2000
EAF: Stainless & Non-Stainless Steel (2)	--	delisted 5/17/96
Ferroalloys	02/98	08/98
Flexible Polyurethane Foam Prod.	12/09/96	03/98
Mineral Wool	04/29/97	04/98
Nylon 6 Production	--	to be delisted
Oil & Natural Gas Production	12/97	10/98
Petroleum Refineries	03/98	03/99
Pharmaceuticals Production	03/20/97	04/98
Polycarbonates Production (GMACT)	01/98	12/98
Polyether Polyols Production	08/15/97	09/98
Polymers & Resins III (3)*	05/98	07/99
Portland Cement	01/98	09/98
Publicly Owned Treatment Works (POTW)	02/98	01/99
Primary Aluminum	08/22/96	09/19/97
Primary Copper	01/98	06/98
Primary Lead Smelting	01/98	08/98
Pulp & Paper (non-combust) MACT I^	12/17/93	11/14/97
Pulp & Paper (combustion) MACT II^	11/14/97	07/98
Pulp & Paper (non-chem) MACT III^	02/29/96	11/97
Reinforced Plastic Composites Prod.	10/99	11/2000
Secondary Aluminum Prod.	03/98	03/99
Steel Pickling	08/28/97	04/98
Wood Treatment MACT	--	delisted 5/17/96
Wool Fiberglass	02/25/97	03/98

Key Legend:

\* = Standards with more than one Source Category (see below for break-down)

^^ = formerly known as Butadiene Dimers Production

^ = projects are part of the Pulp & Paper rule

**Exhibit C.3 (cont.)  
EPA - Clean Air Act - Title III  
7-Year MACT Standards**

7 YEAR STANDARD BREAK-DOWN OF SOURCE CATEGORIES

AGRICULTURE CHEMICALS PRODUCTION:

- 4-Chloro-2-Methyl Acid Prod.
- 2,4 Salts & Esters Production
- 4,6-dinitro-o-cresol production
- Captafol Production
- Captan Production
- Chloroneb Production
- Chlorothalonil Production
- Dacthal (tm) production
- Sodium Pentachlorophenate Prod.
- Tordon (tm) Acid Production

CYANIDE CHEMICALS PRODUCTION:

- Sodium Cyanide Production
- Hydrogen Cyanide Production
- Cyanuric Chloride Production

POLYMERS & RESINS III:

- Acetal Resins
- Amino Resins
- Phenolic Resins

PULP & PAPER:

- MACT I - non-combustion
- MACT II - combustion (kraft, soda, sulfite)
- MACT III - non-chemical
- NESHAP for Combustion Sources in the Semichemical Pulping Industry

**Exhibit C.4**  
**EPA - Clean Air Act - Title III**  
**10-Year MACT Standards**

Statutory date - 11/15/00 (87 Source Categories)

Updated as of October 1997

<b>10 YEAR STANDARDS</b>	<b>PROPOSE</b>	<b>PROMULGATE</b>
Aerosol Can-Filling Facilities	potential	delisting
Alumina Processing	11/99	11/2000
Ammonium Sulfate Production	11/99	11/2000
Antimony Oxides Manufacturing	potential	delisting
Asphalt Concrete Manufacturing	11/99	11/2000
Asphalt Roofing & Processing	08/98	08/99
Asphalt/Coal Tr Application-Metal Pipes	11/99	11/2000
Auto & Light Duty Truck (surface ctg.)	11/99	11/2000
Baker's Yeast Manufacturing	10/98	06/99
Boat Manufacturing	12/99	12/2000
Carbon Black	11/99	11/2000
Carbonyl Sulfide (COS) Production via Carbon Disulfide	11/99	11/2000
Clay Products Manufacturing	11/99	11/2000
Coke By-Products	covered by 40CFR61 Sub L	
Coke Oven: Pushing, Quenching...	11/99	10/2000
Dry Cleaning (Petroleum Solvent)	11/99	11/2000
Engine Test Facilities	11/99	11/2000
Ethylene Processes	11/98	11/99
Flat Wood Paneling	11/99	11/2000
Flexible Poly Foam Fabrication Operations	03/99	06/2000
Friction Products Manufacturing	05/99	04/2000
Fume Silica Production	11/99	11/2000
Hydrogen Chloride Production	11/99	11/2000
Hydrogen Fluoride Production (GMACT)	01/98	12/98
Industrial Combustion Coord. Rule +	11/99	11/2000
Integrated Iron & Steel	11/99	11/2000
Iron & Steel Foundries	11/99	11/2000
Large Appliance	07/99	11/2000
Lead Acid Battery Manufacturing	--	delisted 5/17/96
Leather Tanning & Finishing Operations	11/99	11/2000
Lime Manufacturing	04/99	04/2000

**Exhibit C.4 (cont.)  
EPA - Clean Air Act - Title III  
10-Year MACT Standards**

<b>10 YEAR STANDARDS</b>	<b>PROPOSE</b>	<b>PROMULGATE</b>
Marine Vessel Loading Operations	--	7/28/95
Metal Can	11/99	11/2000
Metal Coil	11/99	11/2000
Metal Furniture	07/99	11/2000
Miscellaneous Cellulose +	12/99	11/2000
Miscellaneous Metal Parts	11/99	11/2000
Municipal Landfills	11/99	11/2000
Misc. Organic NESHAP (MON) +	11/99	11/2000
Nitrile Resins Production ^^	--	05/15/97
Non-Clay Refractories Manuf.	05/99	05/2000
Organic Liquids Distribution (Non-Gas)	11/99	11/2000
Paint Strippers	11/99	11/2000
Paper & Other Webs (Surface Ctg)	11/99	11/2000
Phosphoric Acid/ Phosphate Fertilizers ^	11/21/96	12/97
Plastic Parts & Products	11/99	11/2000
Plywood/Particle Board Manuf.	11/99	11/2000
Polyvinyl Chloride & Copolymers Prod	11/99	11/2000
Primary Magnesium	05/99	05/2000
Printing, Coating, & Dyeing of Fabrics	11/99	11/2000
Quaternary Ammonium Comp. Prod.	11/99	11/2000
Rocket Engine Test Firing	11/99	11/2000
Rubber Tire Production	03/99	12/99
Secondary Lead Smelters	--	5/31/95
Semiconductor Manuf.	11/99	11/2000
Sewage Sludge Incinerators	04/99	05/2000
Site Remediation	11/99	11/2000
Spandex Production	11/99	11/2000
Taconite Iron Ore Processing	11/99	11/2000
Uranium Hexafluoride Production	11/99	11/2000
Vegetable Oil Production	11/99	11/2000

Table Legend:

+ = standards with more than one source category (see attached for breakdown)

^ = two source categories being worked on together as one project

^^=Part of Polymers & Resins IV

**Exhibit C.4 (cont.)  
EPA - Clean Air Act - Title III**

## **10-Year MACT Standards**

### BREAKDOWN OF SOURCE CATEGORIES FOR 10 YEAR MACT

#### MISCELLANEOUS CELLULOSE MACT

- Carboxymethylcellulose Production
- Cellulose Ethers Production
- Cellulose Food Casing Manufacturing
- Cellophane Production
- Methylcellulose Production
- Rayon Production

#### INDUSTRIAL COMBUSTION COORDINATING RULEMAKING

- Industrial Boilers
- Institutional/Commercial Boilers
- Process Heaters
- Stationary Internal Combustion Engines
- Stationary Turbines

#### MISCELLANEOUS ORGANIC NESHAP (MON)

- Alkyd Resins Production
- Benzyltrimethylammonium Chloride Production
- Carbonyl Sulfide Production
- Chelating Agents Production
- Chlorinated Paraffins Production
- Ethylidene Norbornene Production
- Explosives Production
- Hydrazine Production
- Maleic Anhydride Copolymers Production
- Manufacture of Paints, Coatings, & Adhesives
- OBPA/1,3-diisocyanate Production
- Photographic Chemicals Production
- Phthalate Plasticizers Production
- Polyester Resins Production
- Polymerized Vinylidene Chloride Production
- Polymethyl Methacrylate Resins Production
- Polyvinyl Acetate Emulsions Production
- Polyvinyl Alcohol Production
- Polyvinyl Butyral Production
- Rubber Chemicals Production
- Symmetrical Tetrachloropyridine Production